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AUDIOMETRY IN GENERAL PRACTICE

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BOSTON

"THE proof of the deafness is in the hearing," to paraphrase an old adage. Essential as the physical examination of the ear is, it gives no accurate clue to the efficiency of that ear. One is often amazed at the acuity of an ear showing the effects of severe mechanical damage, whereas a person presenting anatomically perfect eardrums may be profoundly deaf. If one adds to this the fact that in by far the greatest number of cases the onset of the deafness is very insidious, one may say that audiometry is of paramount value in the detection of deafness. It is the purpose of this paper to describe some of the various means and methods of testing hearing, especially those that should be incorporated in the daily routine of medical practice.

For the past several years, two types of electric audiometer have been extensively used by otologists, hospitals, laboratories and schools in the detection and the measurement of deafness. The so-called "pure-tone" audiometers produce musical tones of variable pitch and intensity by means of electric oscillators. With their aid, the hearing acuity of an ear can be charted in the form of a curve. Their use requires a good deal of practice and application, especially since, in spite of their much vaunted "scientific accuracy," they do not account for the intangibilities of the human element. Audiometry, it must be remembered, is largely a subjective test unless one resorts to the "conditioned reflex" methods of the animal experimenter. As a matter of fact, in the following paper, Dr. Guilder states that she has successfully used that method in her work on preschool children. Far more applicable in crusade work against deafness are the so-called "group audiometers," which have been used for some years in the public schools of Massachusetts and other states. The group audiometer consists of a phonograph with an electric pick-up to which as many as forty earphones can be connected. The disks used in the phonograph are carefully made recordings of monosyllables or numbers, which the pupils must write down as they hear them. Any pupil whose record sheet shows more than the permissible number of mistakes is then given a note to the parents advising them of the fact that the child has a hearing defect, and that they should consult their family physician, who refers the patient to an otologist or an ear clinic, where more accurate tests can be made. Rough as the group audiometer test, also called "screen test," may be, it is surprising how frequently even very slight losses of hearing are being detected. It can safely be said that in the majority of such cases the hearing loss is entirely unsuspected by parents and teachers alike, to say nothing of the patient. It stands to reason that such a program of deafness detection is of the greatest service to the public, and it is to be hoped that ultimately all the states of the Union will avail themselves of the systematic use of the group audiometer in the public and other schools.

The electric audiometers just described are obviously not adapted for the purpose of the busy general practitioner, who must have more easily available means for testing a patient's hearing. Undoubtedly, the oldest and, for all practical purposes, the most valuable of these is the human voice. The voice test has of late (since the introduction of the electric audiometer) been much maligned as being "unscientific and inaccurate." It must not be forgotten, however, that the human voice is more important to the human ear than any other sound. In general practice, it is much more essential to determine quickly and roughly the hearing acuity for the voice than to measure it "accurately" in terms of cycles and decibels. Two types of voice are usually and properly employed: the whispered and the conversational.

Of the two, the whisper is the more reliable because, as a rule, it is easier to keep it on a fairly even intensity level. A few simple rules should be observed. A "stage whisper" is not permissible.

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In practicing the true whisper, the examiner should place his fingers lightly over the region of his thyroid cartilage; if he can feel the slightest vibration, he is not whispering properly. In the correct whisper, the vocal chords remain comparatively if not actually inactive, the whisper being produced almost entirely by the accessory voice organs such as the lips, teeth, tongue and palate. What vocalization is present is produced by a change of the size and shape of the mouth and pharynx. It has often been suggested that so-called "supplemental air" be used in whispering, and in the conversational voice for that matter, to prevent the whisper from being forced. That bit of technic is excellent but time consuming and tiring. It is suggested that the examiner fix in his mind the intensity of the whisper thus produced and then merely use his air sparingly. With a very little practice, a useful whisper can easily be acquired.

The proper use of the conversational voice involves more difficulties. It is hardly necessary to call attention to the tremendous variations in voices; every day, one listens to voices that boom, squeak, shout and mumble. The examiner should speak as he would in a quiet room to a near-by person with good hearing. Certain precautions must be observed. The ear being tested must be turned toward the speaker, whereas the other ear must be occluded. That is best done by having the patient insert the moistened tip of his little finger into the ear canal; a cotton plug is not sufficient. The relative position of examiner and patient is such that lip reading is automatically excluded. In the ordinary home or office, the conversational voice is not so applicable as the whisper, for an ear must be deaf indeed if it cannot hear and, what is much more important, understand such a voice at, for example, six feet.

A long, narrow hall, although affording greater distances, is not desirable for a voice test, because it is likely to act as a speaking tube. It is best, therefore, to begin with the whisper. The distance at which the normal ear can hear the whispered voice depends largely on the prevailing noise level of the room, but also on its size and shape. To set a standard under every-day conditions would be impossible: every examiner must determine his own standard. The choice of test words should depend on the age of the patient, as well as on his level of intellect. Numbers are perhaps the most frequently used, but they offer the patient too many opportunities for guessing. Monosyllables are employed especially for testing the comparative ease with which the patient can differentiate sounds at varying pitch levels. If, for example, the patient can easily tell the difference between such words as "fat" and "sat," his perception for high tones is evidently good. If he can hear "six" but not "four," his perception for low pitches is poor. In my experience, it has been most practical to read to the patient sentences from a nontechnical book or a newspaper, having him repeat every three or four words. The reading should begin at a distance at which the patient can hear comfortably, and continued while one is slowly walking away from him until his responses begin to be hesitating or inaccurate. Such stock questions as "What is your name?" or "Where do you live?" are too easily guessed and should therefore be avoided.

The watch test has definite limitations. If a patient with otherwise good hearing cannot hear his watch tick, one need not be surprised, for the sound produced by the modern wrist watch is extremely weak, and man normally may begin to lose his perception acuity for high tones as early as the fourth decade of life. It is for these reasons that it is hardly fair to use the watch in testing elderly or even middle-aged patients. Here, again, it can be said that every man must establish his own standards. Tuning forks, sound rods, the Galton whistle and the monochord will not be considered here because their use hardly falls within the province of the general practitioner.

Summary

The value of audiometry is stressed. Electric audiometers are described, with special reference to the phonograph type.

The results obtained in the school tests are usually reliable and should be duly heeded by the family physician.

The voice test, if properly applied, can be very useful and reliable. The watch test should be used with caution.

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HEARING HANDICAPS IN CHILDREN OF TODAY: IMPORTANCE OF CLINIC PROGRAMS FOR THEIR EARLY STUDY AND REMEDIAL GUIDANCE*

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THE health, happiness and welfare of potentially normal children are becoming more and more the concern of otology, as of every medical specialty dealing with the physical well-being of the children of today. Specialties within specialties are being devoted to the study of the relatively normal child at the different age levels, so that defects, which formerly went unrecognized until late childhood or even adult years, may be diagnosed and remedied at the earliest possible moment. Adjustment to handicaps found to be permanent is being pushed down to the youngest possible age, so that the child may have every opportunity of proceeding as a normal or nearly normal person during his school years. Thus, all are working not only for the greater happiness of today's children but also for the greater efficiency of the coming adult generation. Adjustment and compensation, which may be made with comparative ease during childhood, may often be very difficult or even impossible in adult years.

The hearing handicaps of childhood are demanding, as never before, the development of specialized clinic programs for children within the field of otology. With this growing emphasis on the early detection of physical defects in childhood, an increasing number of children at the different age levels, from eighteen months to eighteen years, with hearing impairment varying all the way from the slighter deviations from normal to profound deafness, are coming to the otologist and the ear clinic for help. Some respond promptly to treatment, and therefore call for only a periodic clinic check-up to determine whether the improvement remains permanent. Others present irremediable defects of various types and degrees. It is not surprising that some of the hearing defects of childhood, like the visual defects, should prove to be permanent handicaps, when one realizes that the very delicate nervous mechanism of hearing is likely to be the seat of various degrees of developmental deviation and degeneration, that the auditory nerve has proved to be the most susceptible of all the cranial nerves to traumatic and toxic damage, and that some types of conduction deafness with hereditary backgrounds may have their onset in early childhood as well as at adolescence or in young adult years. Detailed clinic histories are showing that a hereditary background is more frequent than was heretofore realized in the partial hearing defects of childhood. Children who are found to have permanent handicaps continue to remain the problem of the otologist and the ear clinic. It is for these children that clinic programs for individual study and remedial guidance based on such study are essential; with the exception of the very small percentage of the profoundly deaf, they are potentially normal or nearly normal children.

Adequate programs for children with hearing handicaps have been a little slower in developing than similar programs for the other major physical handicaps of childhood, partly because of the greater difficulties of diagnosing and evaluating the handicap at the younger age levels, and partly because until the present time satisfactory mechanical means of improving hearing for the individual child have been lacking. Both these factors have now been largely eliminated. New technics are being developed for the diagnosis of hearing impairment during preschool years, and methods for accurately evaluating the child's hearing ability and disability throughout the years of childhood are being adapted to the different age levels; most important of all, the vacuum-tube hearing aid has at last made it possible to offer better hearing to every child in need of help, provided he has some residual hearing over the speech range. Better hearing to these children is no longer a dream, but a reality. Unaided, they constitute one of the most severely handicapped groups. Adequately studied and rehabilitated at the youngest possible age, they constitute one of the most hopeful of the physically handicapped groups, ready to carry on as essentially normal persons in the classroom and in many occupations and professions when they reach adult years.

The moment has therefore arrived when such a study-service program should be available to every child with a permanent hearing handicap. Such programs should develop ideally in large clinic centers, so that the child may have the benefit of any other examinations or treatments that may be indicated.

To meet the needs of the increasing number of such children coming to the Massachusetts Eye and Ear Infirmary from all parts of New England, the Winthrop Foundation has, as one of its activities, made a small beginning on the development

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of such a program. The Foundation is most grateful for the co-operation of all who have made this beginning possible: the clinic staff, the social-service staff, the three highly trained teachers who volunteered their services so that the technics of therapeutic teaching might be worked out, the several hearing aid firms who have met the special needs of the project so efficiently, and all who have referred these young people to the clinic.

On the basis of experience at the Winthrop Foundation, the following problems are briefly discussed: the gravity of the handicap as it appears in the cross-section of New England children studied; the hearing study and its adaptation to the different age levels; and the program for remedial guidance and its adaptation to meet the needs of the individual child.

Gravity of Handicap

A hearing defect of any degree, even the slighter deviations from normal, may be seriously handicapping in early childhood, because of the vital part that hearing plays in the child's speech-language development, in his early intellectual growth and, later, in his education.

His first efforts at speech are an imitation of what he hears. If the sound of voice fails to reach him, he will not develop speech until taught by visual and kinesthetic methods.* If speech is a blur of confused sounds to him, his first efforts at imitation will be delayed and quite imperfect, but will gradually improve as he learns to listen more carefully. His first words may consist largely of vowel sounds. If, as in children with some of the slighter high-tone deviations, he fails to hear only the soft, high-pitched consonant sounds, these sounds will be added slowly and imperfectly, and he may substitute one consonant for another. Frequently, the speech defect so predominates the picture that the underlying hearing impairment may go unrecognized for a number of years.

The young child with even a slight reduction in hearing ability increases his vocabulary more slowly than his normal playmates, and may seem in various ways retarded because of the restriction of his auditory field, even though he has a good intellectual endowment. The child whose hearing distance is only two or three feet misses all the innumerable opportunities of acquiring new words and new facts that make the young child's mental growth so incredibly rapid. If the hearing distance is increased, an intelligence quotient, based to any degree on an understanding of language, is often raised in a short time.

Too much can scarcely be said about the handicapping effect of a hearing disability of any degree during school years. Such disabilities certainly rank high among the tragedies of school children at present. They are one of the principal causes for the repetition of grades. They are responsible for many of the behavior problems of childhood and adolescence. The child may have been taught to read the lips, but he still loses his place when the children behind him are reciting; he fails to understand the spelling words or the test questions, if the teacher happens to move to another side of the room.

It is therefore apparent that, from the age of twelve months and on through the preschool and school years, a hearing impairment of any degree is a serious disability, and even the slighter ones are far more handicapping than similar degrees of impairment occurring for the first time in adult years. The adult has years of experience in listening, and is often able to supply the word or sound that he does not quite hear, whereas the child is constantly being presented with unfamiliar words and strange new facts.

In addition to the effect of the handicap on early development and education, many pleasures are denied the child with a partial hearing disability—all the soft sounds indoors and outdoors, such as the singing of birds, the chatter of children, the rumble of the city traffic and the sound of a distant airplane. It is hard to forget the eagerness of a little boy who rushed to the door during his first hearing-aid demonstration and stood enthralled at the sound of the cars whizzing by—or of the five-year-old girl who was so surprised at the sound of the chatter of the other children when she first wore her hearing aid to kindergarten.

Hearing Study

To evaluate the child's hearing defect in terms of hearing ability and disability, which may serve as a basis for the clinic remedial program and for interpretation to the family and the teacher, the hearing test has been converted into a hearing study.

For the *child of school age,* one must know not only the character and degree of the hearing impairment in terms of a pure-tone audiometer test by air and bone conduction and of tuning-fork tests, as described by Dr. Mueller in the preceding paper, but also the amount that he is handicapped in hearing of monosyllables, sentences and new school material, such as he might have in school the next day. This estimate is made in two ways: by a phonographic attachment to the audiometer, the amplification necessary in each ear for the child to hear a list of fifty words correctly is determined; and by voice, his unaided binaural hearing for

*This group constitutes a very small proportion of the total number, and is provided for in schools for the deaf.

words, sentences and school material is studied and his hearing distance for different types of material, as well as for individual speech sounds, is established. In this way, one can determine how much difficulty the child has, when he is unable to read the lips of the teacher or of the child who is reciting behind him. All these procedures are necessary, because throughout the years of childhood, there is often not a perfect correlation between the pure-tone test and the interpretation of words heard, which is a function of the higher cortical centers. These various technics are adapted to the age level of the child. Speech defects are recorded as noted. The hearing study is an excellent opportunity for interpretation of the child's difficulty to him and later to his parents.

For the *preschool child*, the entire study is carried out in a nursery-school setting, with play material and with an observation and play-conditioning technic. The child's spontaneous responses to voice, music and a number of gross sounds of different pitches are first studied, and then his trained response, by conditioning him to "pump a ball" or "jump a horse" when he hears a bell or a drum tap. Later, the same procedure is used with the word "pump" or "jump," and in this manner his hearing distance for conversation and whispered voice can be determined, even though he has only a limited understanding of language or none at all. A word-picture game is used for the study of his interpretation of language, and speech is encouraged by play telephone conversations. The audiometer test is introduced as early in the program as possible. A word-picture method is often used up to the age of eight years for study of the child's ability to hear speech.

Remedial Guidance

No one group of the major handicaps calls for more highly individualized remedial programs, based on a detailed study of the child's ability and disability, than the hearing defects of childhood. Yet in no group has the remedial program been more generally routine. The day is past when the routine recommendation of lip reading to every child with a hearing handicap is sufficient. The clinic's duty is not completed at that point: these children deserve the best that medicine and science can give them, and their future happiness and efficiency depend on the proper remedial program at the proper time.

The initial rehabilitation should be considered a therapeutic procedure, growing out of the individual child's needs as evidenced by the cumulative study of his abilities and disabilities in hearing, speech and language during the period of clinic observation, study and therapeutic teaching. The preschool child remains a rehabilitation problem until satisfactorily launched on his school career. The child of school age should remain a clinic rehabilitation problem until he and his parents understand the nature of his handicap and until he has gained self-confidence and become master of the situation by realizing that he is on the road to better hearing. In the clinic, the child is willing to discuss his difficulties, because he comes to tell his troubles, and it is one's responsibility to see that he is equipped to overcome them to the greatest possible degree. In school, he tends to make light of his handicap, and various behavior problems grow out of his effort to cover his real sense of inferiority.

The majority of these children can be given better hearing and, through better hearing, can acquire better speech, better language and an equal opportunity for school progress and for happiness in and out of school. How is one to accomplish this?

When hearing begins to fail, the eye naturally begins to help out the ear by interpreting some speech sounds through watching the movements of the lips. The soft consonant sounds, which are usually the most difficult to hear, are the easiest to interpret on the lips. Therefore, the eye should always be trained to help the ear whenever there is a permanent hearing defect sufficient to interfere with the hearing of speech. Lip reading is thus one of the first of the remedial measures advised, but it should always go hand in hand with the greatest possible use of hearing, so that a hearing aid, as well as lip reading, is advised when the unaided ear cannot easily hear the louder speech sounds. It has been proved conclusively that sight and hearing together give the highest percentage of speech intelligibility at every age level. In addition to these measures, children whose hearing handicap has been present during early childhood need help with speech and language.

For the *slighter hearing defects in children of school age*, lip reading should be recommended if the child's audiogram shows a persistent loss of between 20 and 30 decibels in the speech range, or if his hearing distance for monosyllables, sentences or new school material is less than 20 feet. This will usually be sufficient for the child whose loss occurs at the age of twelve years or over, and he can proceed with the study of lip reading at school, with instructions to return to the clinic for periodic hearing check-ups and further advice concerning remedial measures, if the hearing impairment proves to be progressive. As always, parent and child are made to realize that hearing, like vision, teeth and general health, should be

checked at intervals, that good hearing is one of the most precious endowments and to be carefully guarded, but that the deviations from normal hearing are no rarer than visual defects and that a hearing defect should never be a cause for hypersensitiveness. For children from six to twelve years old, listening exercises on a game basis should be encouraged. Many of these children acquire auditory inattention, because conversation at a distance becomes blurred to them. Parents often describe them as "heedless." They should be advised to make listening fun for the child, and the child himself should be urged to listen carefully.

FIGURE 1.
This photograph shows a group of children having their first training in listening with hearing aids.

For the *hard of hearing and partially deaf group over twelve years of age*, whose audiograms show a persistent loss of more than 30 decibels in the speech range, and whose hearing distance for monosyllables, sentences and new school material is less than 15 feet, a vacuum-tube hearing aid, the study of lip reading and attendance in a clinic class are recommended. In the clinic class, which is taught by a lip-reading teacher, the child has an opportunity to see other children using aids; he gains practice in adjusting his aid and in combining sight and hearing satisfactorily through exercises, guessing games and pleasant conversation. Most important of all, he has a good time, gains self-confidence, and is better able to cope with his handicap. After attending the class for six to eight weeks, he is ready to wear his hearing aid to school and to continue the study of lip reading at school. The class is therefore a revolving one, with a nucleus of old pupils and new ones entering whenever they come to the clinic.

Such a program has proved to be the only satisfactory way of meeting the psychologic and technical problems of adjustment to the new vacuum-tube aids, and the plan is in accord with therapeutic training classes for other types of disabilities in children and young people. Many in the group from twelve to eighteen years are now wearing their hearing aids satisfactorily in school and for outside work and social activities. The majority acquired a hearing distance of at least 20 feet with their aids. In some cases, the remedial program has prevented impending school failure and has made it possible for a high-school boy or girl to go on with his or her chosen line of training. Every effort is made to help this group overcome their hypersensitiveness, and the things to be done to acquire better hearing are constantly stressed, rather than the seriousness of the handicap. As the program for better hearing has grown, parents, children and young people have helped spread this feeling of hopefulness to the newcomers. Despite the combined efforts of the clinic and social-service staffs, however, some parents and some children and young people refuse to accept the idea

of a hearing aid. For them, there are psychologic obstacles that will be overcome only through the further education of the public to the fact that today a large majority of the children with hearing handicaps can be completely rehabilitated, and that a hearing aid is by no means a sign of lowered efficiency or ability.

The *hard of hearing and partially deaf group six to twelve years of age* present more complicated individual remedial problems. Many have developmental, high-tone defects with the characteristic speech defects and varying degrees of retardation in language development. Some have conduction losses going back to early childhood, with a large amount of rapid but imperfect speech. Many of these children were late talkers, and have continued to reproduce speech imperfectly as they have heard it. These children need the stimulus of clinic class work (Fig. 1), as described above, but they also need individual instruction in speech correction, lip reading and listening exercises by a specially trained teacher. After several months of weekly clinic training, both individual and group, these children are ready to wear their aids satisfactorily in school, and should be able to proceed in public school with the help of special lip-reading instruction, which many school systems provide. A number of these children, without the benefit of a hearing aid and the intensive remedial clinic program, would have been obliged to transfer to a special school for the deaf. It is hoped that a considerable group are being completely rehabilitated. This means that some children will be able to remain in their own homes and in regular classes who formerly were transferred to special boarding schools at considerable expense to the State. Such programs, therefore, organized on a larger scale, can be measured in terms of greater happiness to a group of children and parents, greater efficiency in the coming adult generation and an appreciable financial saving to the State.

The *preschool group* present very special remedial problems. The preschool child with only a slight hearing handicap may have very poor speech, and is usually a very poor listener. At certain times during the day, he may seem not to try to listen. Speech may be so blurred and confused to him that he does not make the effort to listen when he is tired. If his hearing distance for familiar words is 10 to 12 feet, he should be trained and encouraged to listen through games and exercises, which he will enjoy. His imperfect speech sounds should be corrected through hearing to as great an extent as possible, and he should have some training in lip reading, but with emphasis always on the combination of sight and hearing. His clinic program should consist of both group and individual work, and the listening exercises and games should also be carried out at home.

For the partially deaf preschool child, who has developed a small amount of imperfect speech and who has a hearing distance of from 2 to 12 feet for familiar words, a hearing aid, in addition to individual training in listening, lip reading and speech correction through hearing, is recommended. Again, the stress is placed on a combination of sight and hearing. The entire program, from the hearing study throughout the training period, is carried out on a play-school basis. The group class often seems like a party to these younger children (Fig. 2). The hearing aid is first introduced as a play telephone, and a word-picture game is used for measuring its usefulness at various distances.

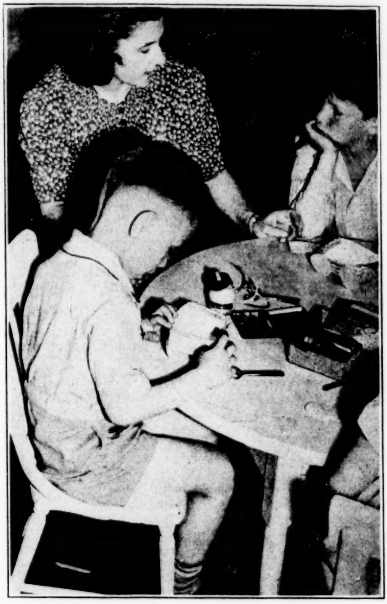

FIGURE 2.

This photograph shows how learning to listen and to lip-read are made fun for young children.

A five-year-old girl has been wearing an aid the entire day for six months. She has worn it in kindergarten as unconsciously as a child might wear a pair of glasses. Her hearing distance has been increased from 2 feet to 15 feet, and she has

lowered the pitch of her voice, corrected her own imperfect speech sounds, and increased her vocabulary from about thirty words to sufficient language to carry on a normal conversation for her age. She is now able to take part in games, whereas formerly she stood on the outside and watched. Being fitted to an aid at just this crucial moment, she should have a normal school career and a much happier, fuller childhood.

If the age of rehabilitation can be lowered to three years, still more of the critical years of early childhood may be retrieved, and speech and language may be acquired more completely in those years of greatest speech readiness. The detection, evaluation and correction of hearing disabilities during preschool years are a major aspect of the entire problem. A year of childhood lost can never be completely regained. The child is normally most ready to acquire speech and language during his second, third and fourth years, and should be ready at six years to grasp new material rapidly through his hearing. More stress should be placed on the hearing readiness of children before they enter school, and greater efforts made to correct, through proper remedial measures, defects that cannot be cured through therapy. As the mother of a five-year-old child said, "Next to complete restoration of hearing, H——'s complete adjustment to her hearing aid is the nicest thing that could have happened to us."

SUMMARY

With increasing emphasis on the early discovery of physical defects in children, a great need is arising in the field of otology for the development of intensive clinic programs for the detailed study of the abilities and disabilities of children with hearing handicaps, adapted to the different age levels, and for the development of programs for therapeutic remedial guidance, based on such individual studies. The majority of children with hearing handicaps are potentially normal, if found early and adequately rehabilitated. Better hearing is now a possibility for most of these children and, because of this, better speech, better language and an equal opportunity for progress in school and for happiness in and out of school.

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THE WORK OF THE BOSTON GUILD FOR THE HARD OF HEARING

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BOSTON

IMPAIRMENT of hearing is a serious physical defect as well as a social, economic and vocational handicap to a vast number of people. Surveys throughout this country indicate that there are approximately 15,000,000 persons at the present time with some hearing deficiency. This figure includes those with only a mild hearing deficiency and a large number of younger persons who readily respond to treatment. Nevertheless, it leaves several million with a permanent and serious hearing loss who cannot be helped medically and who are in real need of readjustment.

Many of these are hopeful cases, owing to the availability of agency services and a better understanding of hearing problems. It is therefore of real value for physicians to know about the local centers that serve deaf people and to become more familiar with the programs that enable the hard of hearing to remedy and surmount their difficulties.

The Boston Guild for the Hard of Hearing, located at 283 Commonwealth Avenue, is a social agency operated by and for the hard of hearing of Metropolitan Boston. It offers its services not only to its members but also to the community by helping the person with a hearing loss to develop his remaining capacities and to make adjustments along a number of concrete lines.

One outstanding activity of the Guild is its hearing-aid guidance service. The first step taken when a person comes for help is to study his particular case. To do this, several pertinent questions are always asked. Has he received adequate care from an otologist and has his hearing been given all the benefit of medical treatment? If not, medical advice is recommended either by an ear specialist of the patient's choice, by one of the Guild's consulting otologists or by the service of a dispensary, as the case indicates. Many persons are referred directly from physicians or hospitals, and in these cases a summary is frequently sent in advance. The second question asked is, What is the degree and character of the hearing loss? If this has not already been determined by an otologist, the Guild is prepared to make an audiogram, using a standard audiometer. If it is found that a person will be benefited by and is able to finance a hear-

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ing aid, the next question is, Which of the several types and makes are most suitable? Laboratory evaluations of these instruments are not available to the average purchaser, but the only instruments in the Guild demonstration collection are those approved by the Council of Physical Therapy of the American Medical Association. It is believed that there is no best hearing aid for everyone, and an hour's comparison of the various instruments is an effective means of deciding which device assists the patient to make use of his impaired hearing to the fullest possible extent.

After one of several hearing aids is selected as giving the best results, the person is advised to consult the company where the instrument may be tried out and purchased. Every year, an increasing number of otologists and general practitioners refer patients to the Guild. This service aids the physician in overcoming the patient's habitual resistance to wearing a hearing aid and is a help to the person bewildered about which instrument to purchase among those so widely advertised.

In addition, the Guild has a few hearing aids that are available as loans to persons who are prevented by hearing difficulties from obtaining and maintaining employment and who have no means of financing an instrument.

Lip reading is another means of rehabilitating the deafened. Almost anyone whose vision enables him to see a speaker's lips at a distance of three or four feet can learn something about lip reading. The amount he learns depends on his attitude toward his handicap, his perseverance and his regular attendance at classes. The slightly hard of hearing can profit fully as much as the severely deafened and should be urged to take up the study of lip reading early, for this skill develops only gradually and with much practice. Hearing-aid salesmen often say that the most successful users of instruments are those who have learned to concentrate their attention on the speaker's mouth as a result of lip-reading lessons. It is therefore essential to stress the study of lip reading as well as the use of a hearing aid. The former also offers a solution to the person who cannot be helped medically and who is unable to hear with an audicle. Lip reading bridges the gap for the person whose loss is not severe enough to warrant the use of a hearing aid.

The Guild is not a school of lip reading, but gives classes in practice and drill, so that anyone who wishes to master the art is urged first to take private lessons from qualified teachers. It has free afternoon and evening classes, however, for those who cannot afford tuition elsewhere.

Another service is conducted for children with hearing difficulties by the giving of audiometer tests. Parents are also advised about adequate educational provisions for the child who is so handicapped. Always alert to outside contacts, the Guild interests school and health authorities in hearing tests for school children so that preventive and helpful measures may be taken in time. It owns a 4C audiometer, especially designed for the testing of school children in groups of as many as forty, which is available to any public or private school on the payment of a small upkeep charge. A trained audiometer technician on the Guild staff is also available to do this testing in the schools.

Massachusetts has its own department of vocational rehabilitation and a division for the hard of hearing under a trained social worker, who cooperates with the Guild in securing retraining and employment for many people.

The Boston Guild is also a service club offering a varied social and educational program to its members. This was founded on the premises that the isolation of the hard of hearing is largely responsible for traits often attributed to them — sensitiveness, suspicion, lack of courage and initiative — and that the cure lies in the removal of the cause. Entertainments, lectures, card parties, teas, out-of-door trips and gatherings for various other purposes provide incentive for people who are hard of hearing to get together and make new friendships. Special facilities, including a group hearing aid in the auditorium at the Guild House, make the programs easily available for the members. Men are encouraged to join a very active men's club, and young persons find worth-while outlets in the young people's group.

The Guild also offers opportunity for growth in personality and creates normal outlooks with the right perspectives on impaired hearing. Otologists with patients who are isolating themselves and who need a better attitude toward their handicap would do well to recommend membership in the Guild. Those who wish this privilege, however, should not belong to the deaf group, since facilities for them are provided elsewhere. Nevertheless, any deafened person may make formal application for membership at the Guild if he furnishes the necessary references.

Many people with normal hearing, among them physicians, not only support the Guild's work but become members. In no other way can they understand the urgent problems of hearing that are rapidly becoming more critical in modern society.

The Boston Guild for the Hard of Hearing is supported by membership dues, by rent from rooms

in its house, by voluntary contributions from members and friends, by interest from endowments and by participation in the Greater Boston Community Fund campaign.

During National Hearing Week, the last full week in October, the local chapters bring their work and that of the American Society for the Hard of Hearing to the attention of the general public by radio talks, lectures, pamphlet material, posters, demonstrations of lip reading and publicity of various kinds. Otologists and physicians are welcome to any of these activities.

There are eleven leagues for the hard of hearing in Massachusetts and thirteen in four other New England states, all of which are constituent chapters of the American Society for the Hard of Hearing and work actively in the interests of persons so afflicted.

283 Commonwealth Avenue

CASTRATION FOR CARCINOMA OF THE PROSTATE*

Report of Forty-One Cases

C. H. Neuswanger, M.D.,† and Vincent Vermooten, M.D.‡

NEW HAVEN, CONNECTICUT

THE report of the effect of castration on carcinoma of the prostate by Huggins et al.§ before the meeting of the American Urological Association in June, 1941, at Colorado Springs so impressed us that we have urged all our patients with carcinoma of the prostate to permit a bilateral orchidectomy. To date, we have operated on 41 of these patients, none of whom, up to the present, have regretted having the operation done. One patient died of pneumonia five months after operation. In the remaining cases, there are no complaints. On the average, the patients gained a minimum of 5 pounds in weight, and many as much as 20 or 30 pounds. The most striking changes observed were a marked increase in appetite, complete relief of pain, improvement in bowel habits and a feeling of well-being. There was also complete relief of urinary symptoms, but all patients except 1 had also had a transurethral resection of the prostatic carcinoma, which, no doubt, contributed considerably to this improvement as well as affording an opportunity to confirm the clinical diagnosis.

Our observations substantially confirm those reported by Huggins and his co-workers, even to the almost miraculous response of one patient who had a paraplegia resulting from a metastatic lesion in the spine.

In the following cases, the results were striking.

Case Reports

Case 1. G. A. P., when first seen in March, 1940, was found, on rectal examination, to have a carcinoma of the prostate. Except for nocturia once and some slight day frequency, he had no symptoms of note. In May, 1941, the nocturia had increased to three or four times, and the frequency during the day to once every hour or so. The patient was not seen again until the end of September, 1941, when he had developed complete retention of urine, with overflow incontinence. He was immediately admitted to the hospital.

Rectal examination showed a huge prostatic carcinoma, which had occluded the urethral lumen to such an extent that it was not possible to pass even a filiform guide through it. Bilateral orchidectomy was done the next morning, no attempt being made to relieve the urinary retention.

Within 48 hours, the patient was voiding small quantities spontaneously, and by the end of a week, the bladder, which had previously been distended to the umbilicus, could not be felt or percussed above the symphysis. At present, the prostate is greatly reduced in size, although not yet within normal limits. The patient is able to empty his bladder completely and voids only twice at night. He has gained 22 pounds in weight and is symptom free.

Case 2. L. J., a 77-year-old man, was seen during the latter part of November, 1941, complaining of priapism, which had begun about 3 months previously and was continuing for longer and longer periods without relief. He had no other symptoms or discomforts except that he claimed that intercourse made the priapism worse and more painful.

Rectal examination revealed a large, hard, nodular, carcinomatous prostate.

Following a transurethral resection (to obtain tissue for microscopic examination) and a bilateral orchidectomy, the priapism gradually subsided until the 4th postoperative day, when the penis was completely flaccid, in which condition it has remained ever since.

Case 3. D. M. D., a 76-year-old man, was admitted to the hospital for relief of respiratory distress in September, 1941. In March, 1936, one of us (C. H. N.) had operated on him, doing a radical prostatectomy and seminal vesiculectomy for carcinoma. Since that time, he had been considered cured. It was learned, however, that owing to marked respiratory distress he had been bedridden for 3 months.

On admission to the hospital, the patient was practically moribund. X-ray examination revealed that the lung fields were riddled with metastatic carcinoma, as were the bones of the pelvis, the lumbar and dorsal portions of the spine and the ribs. The patient had no urinary symptoms, and there was no local recurrence of the carcinoma.

*Presented at a meeting of the New England Section of the American Urological Association, Boston, February 12, 1942.

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‡Assistant clinical professor of urology, Yale University School of Medicine.

§Huggins, C., Stevens, R. E., Jr., and Hodges, C. V. Studies on prostatic cancer. II. The effects of castration on advanced carcinoma of the prostate gland. *Arch. Surg.* **43**:209-223, 1941.

Since it was assumed that the metastatic growths were from the old prostatic carcinoma, a bilateral orchidectomy was performed under local anesthesia. The general condition rapidly improved, so that the patient was able to go home at the end of 2 weeks. Five months after operation, he was up and about most of the day, doing odd jobs about the farm. His general health was greatly improved, and the respiratory distress almost gone.

Case 4. W. W., a 55-year-old man, was first seen on December 1, 1941, complaining of girdle pains, which radiated from the back around and forward to the umbilicus, of 9 months' duration. These pains were noticed whenever he bent forward or stooped to lift anything. Soon, he noticed that the pain was more marked along the lower costal margin, and he began to have aches and pains in the arms and shoulders as well. He thought this was due to nervousness and worry as the result of the death of his son. On the advice of a friend, he consulted a chiropractor, who manipulated him for one course of thirty-five treatments and a second course of twelve, during June, July and August. Since he did not get a great deal of relief, he was advised to have his teeth extracted. In September, he was sure it was "all nerves" and therefore consulted a neurologist, who had him admitted to the hospital for examination. Neurologic examination was essentially negative, but x-ray films revealed several dental-root abscesses and "widespread osteoplastic changes throughout the spine, pelvis and bony thorax, probably metastatic from a carcinoma of the prostate." After having all his teeth extracted, the patient was sent home with the advice to his physician to give him what sedatives he might need.

Early in October, he began to notice frequency of urination and a month later found that he was not able to use his legs very well. He staggered when walking, and his legs felt weak. This condition gradually got worse until November 15, when, on returning home, he found that he was unable to climb up stairs. He was put to bed and soon discovered that, although he had a great deal of pain and tenderness along the lower rib margin, he had no feeling whatsoever from the waist down. He was unable to move his legs, which "felt dead," and when he attempted to move them his wife noticed that they were "quite stiff." The next day, he was unable to urinate and therefore called his physician, who catheterized him. This was done twice a day until admission to the hospital on December 2.

On entry, the patient had a spastic paraplegia, with complete loss of sensation to pinpricks from the waist down. There was a band of hyperesthesia just below the costal margin, and he was unable to distinguish between heat and cold on the left side.

On December 3, a transurethral resection of the prostate and a bilateral orchidectomy were done. The catheter was removed on the 4th day after the resection, and the patient was subsequently able to void spontaneously. The bowels moved voluntarily, whereas before operation they moved only with an enema.

Two days after orchidectomy, the patient noticed involuntary twitching of his legs, with occasional jerking movements. A week after operation, there was a beginning return of sensation and some slight voluntary movement of the legs. Two weeks after operation, he had almost complete return of sensation from the waist down, and the area of hyperesthesia had practically disappeared. He was also able to move his legs freely in bed. A week later, he was up and walking about with crutches and had complete urinary and bowel control.

When seen on February 10, 1942, the patient had gained 25 pounds in weight. He was able to walk about wherever he pleased with the help of a single cane, which he considered unnecessary. He had no pain or discomfort anywhere, even on bending. He emptied his bladder completely, voided once during the night and once every 3 or 4 hours during the day. Neurologically, we could determine no sensory changes in the lower extremities. The muscle tone was good, and his knee jerks were equal and active. A mass of supraclavicular lymph nodes on the left, which were large, hard and readily palpable when first seen in December, are now definitely much smaller, and the prostate is considerably reduced in size. Microscopically, the tissue resected was reported to be adenocarcinoma of the prostate, Grade II.

Comparing the x-ray films taken 2½ months before orchidectomy with those taken 2 months after operation, one sees that all the lesions became much denser, giving the impression that the disease had become more widespread despite the very marked clinical improvement.

Summary

A report is given on the early postoperative effects of bilateral orchidectomy and transurethral resection on 41 patients with carcinoma of the prostate. Four very striking cases are reported in detail.

PREMONITORY SYMPTOMS OF MYOCARDIAL INFARCTION*

NORMAN H. BOYER, M.D.†

BOSTON

SINCE 1912, when Herrick[1] first described the characteristic signs and symptoms associated with infarction of the myocardium, atypical cases have been recognized with gratifying frequency. Knowledge of the pathology, physiology and pathologic physiology of the coronary circulation continues steadily to advance. The treatment of myocardial infarction, once it has developed, is better understood and more generally applied. Knowledge has apparently progressed to the point where it may now be profitable to turn attention toward recognition of the premonitory symptoms of myocardial infarction in the hope that the institution of therapeutic measures may diminish the severity of the illness or even, in some cases, prevent clinically recognizable infarction entirely.

In a certain proportion of cases, probably a larger number than has been suspected, the onset of myocardial infarction is not sudden and unpredictable but is preceded, for a variable time, by premonitory symptoms, which should receive more serious and general attention. The following cases will serve to indicate some of the danger signals of impending myocardial infarction. Some of the cases furnished a hopeful hint that favorable effects may be expected by prompt recognition and institution of measures designed to increase the absolute or relative coronary blood flow.

CASE 1. A 58-year-old man had noted slight exertional dyspnea for about a year. For 3 months, he had had mild substernal pain, usually after meals. He then began to have attacks of severe, crushing substernal pain, not related to effort and lasting 2 or 3 hours. He had four such attacks in the course of a week. He was seen by his physician several times during that week, and each time the electrocardiogram, white-cell count and erythrocyte sedimentation rate were within normal ranges. Other studies revealed no extracardiac cause for the pain. The patient was advised to go to Florida for a vacation, but 2 days later, he developed a severe attack of pain lasting 8 hours. With this attack, he went into collapse and remained in "shock" for 3 days, with oliguria and rising blood nonprotein nitrogen. The development of fever, leukocytosis (a white-cell count of 18,000) and characteristic changes in the electrocardiogram confirmed the diagnosis of acute myocardial infarction. The course was somewhat stormy, with paroxysmal auricular fibrillation and at least one splenic infarct. However, after 8 weeks in bed and 2 months more of easy convalescence, the patient made a good recovery and has been well and active for the past 2 years.

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In this case, the significance of the repeated prolonged attacks of chest pain during the week preceding acute infarction was not at the time fully appreciated. The duration of the pain seemed to the physician in charge to be too long for angina pectoris, and the absence of fever, leukocytosis and electrocardiographic changes apparently eliminated infarction. It was concluded, therefore, that the heart was not the seat of the pain. Had more attention been paid to the preceding story of substernal pain after meals, it is likely that the prolonged attacks would have taken on new significance. Prolonged substernal pain, with or without characteristic radiation and with or without antecedent angina pectoris, should, in the absence of other obvious causes, be regarded as due to sudden narrowing or closure of a coronary artery. Care must be taken, of course, to avoid confusion with acute coronary insufficiency induced by tachycardia or some other cause.

It is still not widely enough appreciated that the clinical evidence on which proof of the diagnosis of acute coronary occlusion depends consists in manifestations of actual infarction of the muscle. Coronary occlusion and myocardial infarction are not synonymous terms; either may occur without the other, but it is only infarction that produces fever, leukocytosis, changes in the electrocardiogram, friction rub and so forth.

CASE 2. A 58-year-old man, whose family history was unfavorable inasmuch as his mother had died of angina pectoris at 53 and his father of coronary occlusion at 55 years of age, suddenly developed, on March 4, 1941, discomfort over the precordium, with radiation to the back and to the neck. On one or two previous occasions, a similar but less severe and less prolonged pain had occurred. The patient was admitted to the hospital within an hour of the onset, but the pain persisted for several hours despite bed rest and morphine. Physical examination was negative except for occasional premature beats. During the first day in the hospital, two intramuscular injections of papaverine hydrochloride, ½ gr., and atropine sulfate, 1/100 gr., were given at four-hour intervals. Two days later, the pain returned for a few moments following a bowel movement. The patient was kept at rest in the hospital for 2 weeks, during which there was no fever, leukocytosis or increase in sedimentation rate, and no abnormalities or serial changes in the electrocardiogram. He continued to rest at home for another 3 or 4 weeks and has been well and active for the past year.

This appears to be a case of sudden coronary narrowing or occlusion without definite clinical evidence of infarction. Such cases are being recog-

nized with increasing frequency. It seems wise under such circumstances to put the patient at rest, to keep the needs of the heart muscle at an absolute minimum. The use of papaverine is based on its apparent good effects when there is sudden closure of a peripheral artery or embolism to a pulmonary artery. There is evidence that sudden closure of an artery to any part is often accompanied by reflex vasoconstriction of other vessels to that part. Such reflex vasoconstriction in the heart might well interfere with the establishment of collateral circulation, most if not all of which comes from the unoccluded coronary vessels.[2, 3]

The presence of reflex constriction of the coronary bed following ligation of one of the coronary arteries has been indicated by the experimental studies of Manning, McEachern and Hall[4] and LeRoy and Snider.[5] The latter authors were able to reduce the mortality of coronary-artery ligation in dogs by the use of atropine, which they believe interrupted the efferent pathway of the reflex arc. McEachern, Smith, and Manning[6] were able to show a moderate reduction (from 75 to 50 per cent) in the immediate mortality of coronary-artery ligation following the administration of papaverine in doses rather larger than are ordinarily employed clinically.

Not only must one be alert to the possibility of occlusion or narrowing of a coronary artery, without clinically demonstrable infarction on the corresponding myocardium, but also one must guard against haste and impatience in the management of such patients. Failure to provide an adequate period of rest may result in delayed infarction, such as presumably occurred in the following case. It hardly needs to be emphasized that the error of ascribing to the heart pain that belongs in the domain of surgery should be scrupulously avoided. A careful history and good judgment go far toward preventing such an error.

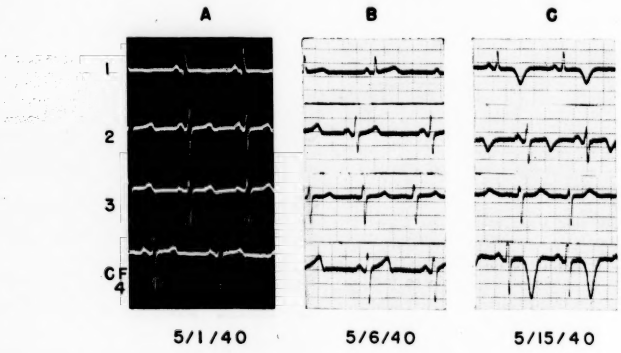

FIGURE 1. *Case 3.*

Record A *was taken twelve hours after a severe attack of substernal pain lasting several hours; there is a considerable degree of left-axis deviation, slight inversion of* T_1 *and an upright* T_3*, with a slightly high origin of* ST_3*; the record was interpreted as indicating left ventricular strain. Record* B*, taken five days later, shows slight changes in the T waves and ST segments; the record is not definitely abnormal. Record* C *was taken fourteen days after admission and the day following a trip to the X-ray Department for a gastrointestinal series; the tracing shows marked inversion of the T waves in Leads 1, 2 and 4.*

CASE 3. A 61-year-old woman with known hypertension for 12 years had been well except for slight exertional dyspnea until 2 weeks before admission to the hospital, when she developed burning substernal pain on effort and after meals. A week before admission, the pain increased in frequency and severity and even came on at rest on one occasion. The day before admission, the patient walked a mile to her doctor's office and on the way home developed a severe substernal pain, which gradually increased in severity and radiated to the arms. The pain lasted several hours, was only partially relieved by nitroglycerin, and was accompanied by weakness and sweating. On admission, the patient had no pain or other complaints. The blood pressure was 194/90. The heart was regular, the sounds were of fair quality, and there were no murmurs or gallop. The lungs were clear, and the abdomen

and the extremities normal. The temperature was normal, and the white-cell count was 10,000. The sedimentation rate, however, was elevated to 1.1 mm. per minute. The patient remained free of symptoms, fever and leukocytosis for 2 weeks. The electrocardiogram on admission showed slight inversion of T_1 and upright T_3 (Fig. 1). The pattern was thought to indicate "left ventricular strain." Five days later, T_1 had become upright. At the end of 2 weeks, the patient was sent to the X-ray Department for a gastrointestinal series because of the absence of any clear indication of myocardial infarction and because of the previously mentioned relation of the pain to meals. Following her return from x-ray study, she developed more pain, the white-cell count subsequently rose to 13,600, and the electrocardiogram showed deep inversion of T_1, T_2 and T_4. There was gradual evolution of the electrocardiogram toward normal in the course of several weeks.

The patient made a good recovery but succumbed to another myocardial infarction 1½ years later.

It seems fairly clear that this patient had coronary occlusion, following a short period of angina pectoris, but without definite evidence of infarction of the heart at the time of admission to the hospital. The untimely trip to the X-ray Department may have precipitated actual infarction of the heart by increasing metabolic demands at a time when the coronary blood flow could support resting needs only. The possibility of a sudden, unrelated occlusion cannot, of course, be summarily dismissed.

Case 4. A 51-year-old man had always been well and very active. In December, 1941, he developed a mild sense of constriction beneath the sternum while riding horseback. The discomfort lasted a few moments. On two or three subsequent occasions, mild substernal discomfort recurred. On February 16, 1942, he developed a severe attack of chest pain lasting several hours. Fever, leukocytosis, and characteristic electrocardiographic changes established the diagnosis of acute infarction.

This case illustrates the fact, now fairly widely known, that myocardial infarction may appear soon after the onset of angina pectoris.

It seems reasonable to suppose that decreasing the demands on the heart, by bed rest during the critical period when collateral circulation is presumably being established, may have a salubrious effect. The possibility that this may occur is indicated by the following case.

Case 5. A 69-year-old man had always been well and active until 10 or 12 days before being seen, when he began to have attacks of substernal pain that were brought on by walking and relieved by rest. Later, the pain was induced by eating. On the day of the examination, the pain appeared following lunch and was partly relieved by nitroglycerin, but the patient still had some pain when seen 2 hours later. Physical examination and fluoroscopy of the heart showed no abnormalities. The temperature was 99°F., and the white-cell count 10,000. The electrocardiogram showed depression of the ST segments in all leads (Fig. 2), a common pattern during spontaneous or induced attacks of angina pectoris.

The patient was hospitalized and kept at bed rest for 3 weeks, during which there was no fever, leukocytosis or increase in the erythrocyte sedimentation rate. The electrocardiogram taken 3 days after the first record showed no ST displacement. Another record a week later did, however, show changes in T_1 and T_3, suggesting that the heart had not entirely escaped damage.

There is, of course, no proof that this patient would not have done equally well if allowed to continue with his usual mode of life, but this seems an entirely unreasonable assumption. Experience with other patients who have suffered extensive infarction of the heart soon after the sudden onset of angina pectoris or following prolonged pain (for example, Cases 1 and 4) leads one to expect a similar course in this patient. That such an infarction did not develop can be fairly attributed to lessening the demands on the heart by means of bed rest.

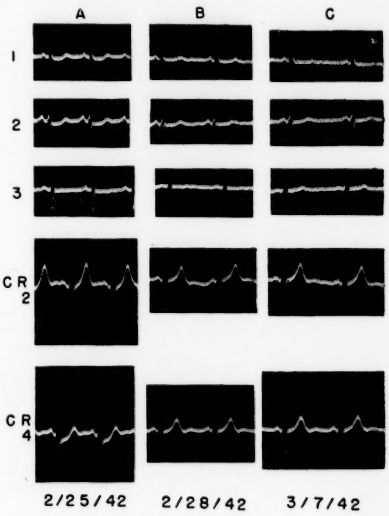

Figure 2. *Case 5.*

Record A was taken during an attack of pain lasting two or three hours; there had been sudden onset of angina pectoris ten to twelve days previously; the record shows depression of the ST segment in all leads. Record B, taken three days later, shows no definite abnormalities. Record C, taken seven days later, shows flattening of T_1 and an increase in the height of T_3, suggesting that there had been some change in the heart muscle.

Case 6. A 60-year-old man, who had had angina pectoris for 5 years, had a sudden increase in frequency and

severity of attacks for 2 or 3 weeks. During this time, the attacks occasionally came on at rest. There was no dyspnea or other cardiovascular symptoms. Physical examination and fluoroscopy of the heart showed no abnormalities. The electrocardiogram showed prolonged auriculoventricular conduction (a PR interval of 0.22 second) and right bundle-branch block. Because of the recent onset pectoris, and at that time the electrocardiogram was normal. In January, 1941, he again complained of chest pain not characteristic of angina pectoris. The electrocardiogram had changed, however, T_1 being inverted (Fig. 3). The patient was advised to curtail his activities, and after a few weeks the pain disappeared. In December, he was seen again because for the previous week he had had dull

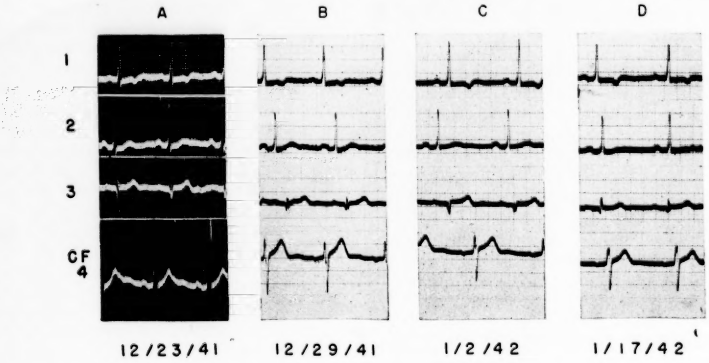

FIGURE 3. *Case 7.*

Record A, *taken one week after the sudden onset of angina pectoris, is identical with one taken eleven months previously; the patient was admitted to the hospital at once because of the symptoms of coronary insufficiency; the record indicates left ventricular strain but not acute infarction. Record* B, *taken six days later, shows no essential change in the T waves or ST segments; the PR interval, however, has lengthened from 0.18 to 0.22 second. Record* C, *taken four days later, shows no change. Record* D *shows slight flattening of the T waves in Leads 2 and 3; the slight T-wave changes and prolonged auriculoventricular conduction suggested that active changes had taken place in the heart.*

of increased coronary insufficiency, the patient was advised to go to bed. This he declined to do. Ten days later, he was seized with a severe attack of chest pain lasting 6 or more hours. Serial changes in the electrocardiogram, a temperature of 101°F. and a white-cell count of 18,000 established the diagnosis of infarction. There was marked gallop rhythm, accentuation of the pulmonic second sound, Cheyne–Stokes respiration and persistence of a shocklike state for 2 days. There was an attack of tachycardia, probably of ventricular origin, although there was no graphic proof. After 3 months, the patient was able to undertake moderate activity without symptoms.

Not only the sudden onset of angina pectoris in a patient previously well but also the sudden increase in frequency and severity of pre-existing angina may presage the onset of myocardial infarction. It is reasonable to suppose that reducing the metabolic needs of the heart during this period may avoid such a critical illness as this patient went through, either by preventing infarction or by materially reducing its size.

CASE 7. A 60-year-old man had complained of vague chest pain in 1937. This was thought not to be angina pain beginning at the back of the shoulders and radiating beneath the sternum. This time, the pain was definitely brought on by exertion and relieved by rest. The electrocardiogram was unchanged from that taken 11 months previously. Physical examination was negative. The patient was put to bed for 2 weeks, and on being allowed up, he developed a moderately severe attack of dull substernal pain lasting 10 to 15 minutes. He was put back at bed rest for another week and then gradually allowed up, without any recurrence of pain. At no time during his hospital stay did fever, leukocytosis or increased sedimentation rate develop. The electrocardiogram, however, showed prolongation of auriculoventricular conduction and slight changes in the T waves, suggesting, as in Case 5, that the heart had not entirely escaped damage.

This is another case in which it is difficult to prove that anything was accomplished by enforced bed rest. The changes in the electrocardiogram indicate, however, that changes were going on in the heart, and it is certainly not unreasonable to believe that such changes were less extensive than they would have been were the patient actively engaged in his usual occupation during this period.

Discussion

The presence of prodromal symptoms probably depends on the rate at which the occlusive process in the coronary vessels develops. Sudden complete closure of a large or moderate-sized vessel will undoubtedly result in immediate infarction of the corresponding myocardium. Atherosclerosis of the coronary arteries is a progressive disease, which is fortunately accompanied by the production of collateral circulation when the need arises. At times, the growth of collateral circulation is unable to keep pace with the progress of the disease, and when the disease is in the ascendancy, symptoms appear or increase. When symptoms are present, indicating that the disease is progressing rapidly, such as the sudden increase of angina pectoris and the onset of prolonged pain, it seems only reasonable to keep the metabolic demands of the heart at a minimum by strict bed rest. In all ischemic tissue, there must be a peripheral zone in which the balance of supply and demand of blood is at a critical point. A little more or less blood or a little more or less demand may mean the difference between viability and death of the tissue. Thus, one might at least expect that, even if infarction occurs, its size may, in some cases, be reduced by virtue of reduction in the demands of the peripheral zone under conditions of absolute rest. If the occlusive process develops slowly enough or if the area affected is small, necrosis of the muscle may not occur. There would seem, therefore, to be little room for doubting the advisability, on theoretical grounds, of minimizing the demands on the heart at a time when symptoms suggest that its blood supply is inadequate, but whether or not any tangible benefit accrues is difficult to evaluate.

Sampson and Eliaser[7] have recorded their experience with the frequency of prolonged attacks of pain, preceding by an interval of days or weeks the onset of infarction. They observed premonitory attacks in 29 patients. Seven patients who were put at bed rest developed infarction despite this precaution, and the mortality rate was not significantly different from that in the remaining patients, who were allowed up and about during the interval between the onset of the premonitory pain and the onset of the infarction. However, they emphasized that the series is too small to allow any conclusions on this point. They had observed 6 other patients, with a single prolonged attack of pain, who were put at bed rest and subsequently did not develop myocardial infarction. They were unwilling to believe, without further proof, that infarction had actually been forestalled. It is true that convincing proof is difficult to obtain, but cases such as Cases 5 and 7, in which slight changes occurred in the electrocardiogram, indicate that changes were occurring in the heart muscle, and it is not unreasonable to assume that such changes might have been more extensive had the demands on the myocardium been greater during this critical period.

Feil[8] also calls attention to the frequency of preliminary pain in myocardial infarction. He states that the pain recurred for an interval of from twelve hours to four weeks preceding infarction, and that he encountered it in 50 per cent of patients with myocardial infarction. He further suggests the point, which needs strong emphasis, that *a normal electrocardiogram does not rule out a developing infarction*. He recommends moderate restriction of the activities of patients during the stage of preliminary pain, but, in view of the gravity of the situation, this seems an inadequate compromise. Complete bed rest under these circumstances has also been urged by Blumgart, Schlesinger and Zoll.[9]

The enforcement of two to four weeks in bed for the patient in whom there is no proof of impending infarction, or proof that the patient will benefit in any way from a period of enforced rest may seem to be radical treatment. Balanced against the possible death of the patient from acute infarction, a prolonged illness extending from three months to a year or more, chronic invalidism, or, at best, a heart with a large myocardial scar, two or three weeks in bed, *if there is the least hope of preventing these sequelae*, seems conservative treatment.

The length of time required in bed varies from case to case. There is experimental evidence that effective collateral circulation requires from one to three weeks for its development.[10, 11] Thus, as a starting point, two weeks should be considered the minimum length of time. It can then be lengthened, if necessary, depending on the patient's response to gradually increasing activity.

It is not suggested that all patients with angina pectoris need be put at bed rest, although even here there is evidence that a period of bed rest at the start of treatment of all patients with angina pectoris may do great good.[12] It is when symptoms of a sudden increase in coronary insufficiency develop that enforced bed rest is urged.

Emphasis has been placed on bed rest, but it is clear from the report of Sampson and Eliaser that rest alone does not prevent infarction in all cases. Other general measures should not be neglected. Primary among these are sedation and avoidance of tobacco. Cognizance should be taken of the increase in cardiac work incident to large heavy meals, and the dietary should be arranged around light foods in small amounts. Drugs al-

leged to dilate the coronary arteries occupy a position of secondary importance. There are reasons for believing that the stimulating effect of the xanthines may be undesirable.[13] Papaverine may be used for its possible effect on overcoming spasm in unaffected vessels that serve as the source of supply for the collateral circulation. Although it has been shown that the maximum collateral flow occurs only after several days or weeks, an immediate increase,[10] although slight, may conceivably keep all or part of the ischemic myocardium viable if such collateral flow is not hampered by reflex constriction of the vessels that provide the source for collateral circulation and if, at the same time, the metabolic needs of the heart are minimized. Atropine may also be used on the basis of previously cited experimental work, although the tachycardia incident to its use may not be altogether desirable. So far as is known, papaverine has no undesirable effects on the heart.

Summary and Conclusions

Sudden onset of angina pectoris, sudden increase in the severity and frequency of pre-existing angina pectoris, and prolonged chest pain without signs of cardiac infarction have been found to precede the onset of infarction in some patients.

Recognition of these warning symptoms and proper treatment by rest, sedation, avoidance of tobacco and the use of papaverine may, in some cases, reduce the size of the eventual infarction or possibly forestall it entirely.

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PROTEINURIA AND THE ASSOCIATED RENAL CHANGES*

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THE first observers of proteinuria, Dekkers (1694) and Cotugno (1765), theorized that it should occur in certain conditions, made a single observation that happened to verify their erroneous theories and thus missed the true significance of the phenomenon.[1] Cruikshank, Wells and Blackall[2] first made systematic studies, and by 1827, John Bostock,[3] studying his own cases and those of Bright, had arrived at the main truths known today: albuminuria occurs in many circumstances, often "of a trifling nature"; the protein in the urine "possesses every property of that in the blood"; the specific gravity of urine from cases of chronic proteinuria is "less than that of the urine in the healthy condition of the system"; "the serum generally in these cases contains less albumin than in health"; and "the blood exhibits a very great deficiency of albumin at the same time that we observe the mode by which it passes off from the system by means of the kidney."

The large, greasy yellow kidney found in many cases of Bright's disease, with edema had tubular changes characterized by desquamation, regeneration with mitosis and large amounts of lipoid in the cells of the tubules. The changes seem not unlike those in the heart and brain injured by anemia, or in the liver injured by toxins or anoxia. Pathologists were impressed by the severity and constancy of the tubular changes, which they regarded as probably toxic in origin. They even suggested that proteinuria might be due to tubular injury or be produced by tubule cells, as mucus is thrown off by injured cells in the nasopharynx, bronchus or colon. Modern methods such as ultracentrifugation and electrophoresis,[4, 5] and also specific biologic tests show that the albumin and globulin, which are always found together in proteinuria, are identical in their properties with molecules of protein in the plasma and are not to any appreciable degree derived from the tissues. Studies of fishes with aglomerular kidneys and of others with glomeruli have shown that without glomeruli

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there can be no proteinuria,[6] and protein can easily be demonstrated in the fluid about glomeruli in cases of proteinuria.[7] Finally, staining methods developed at the Mallory Institute of Pathology, of the Boston City Hospital, have made it possible to show that demonstrable glomerular lesions occur in almost all cases of chronic albuminuria.[8, 9] The problem of proteinuria therefore concerns, primarily, altered permeability in the glomerulus and, secondarily, the effects of this leakage.

Since much information is at hand on the functions of the kidney and on glycosuria and hemoglobinuria, a review of these facts may be useful. By measurement of the ratio of the amount of substance eliminated in one hour's urine to the amount of 1 cc. of plasma (Addis ratio, renal clearance), much has been learned about human renal function. Diodrast clearances are very high, for the tubules and glomeruli eliminate over 90 per cent of the substance as it passes from renal artery to renal vein; this gives a measure of renal blood flow — about 1500 liters a day for a man of normal size. Inulin and sucrose seem only to escape in the glomerular filtrate and to be but little reabsorbed by the tubules; clearance of inulin gives a measure of glomerular filtrate — about 180 liters a day for a man.[10] Richards[11] and his co-workers have analyzed glomerular filtrates from mammals and amphibia, finding the glucose, as well as the chloride and urea, similar to that in the blood, whereas protein, if present at all, is only in minute traces, less than 30 mg. per 100 cc. All the glucose is reabsorbed so long as the plasma level is below 180 mg. per 100 cc., and the reabsorption occurs in the proximal convoluted tubules.[12] Above that threshold, glucose appears in the urine, and above 250 mg. per 100 cc., practically the entire excess escapes in the urine, whereas glycogen appears in the tubule cells in demonstrable masses.

Since hemoglobin has 97 per cent the molecular weight of serum albumin and is tagged with a prosthetic group that makes it easy to study, the investigator of albuminuria is naturally interested in the precise studies of Whipple and his co-workers.[13–17] Unlike Diodrast and neutral red, but like glucose, hemoglobin escapes only through the glomeruli; it is not taken from the blood and excreted by the tubule cells. Animals with no albuminuria show hemoglobinuria only when the plasma level exceeds 100 mg. per 100 cc. However, after prolonged hemoglobinuria, this threshold drops about 60 per cent. The actual leakage through the glomerulus represents only 2.5 to 3.0 per cent of the hemoglobin present in the plasma "cleared" of inulin in the same period. When the blood level is over 100 mg. per 100 cc., hemoglobin and hemosiderin appear in the cells of the proximal convoluted tubule and even farther along in the nephron. Thus, reabsorption, threshold, and tubular storage and transformation occur with this protein, as they do with the carbohydrate dextrose.

Dawson,[18] Hayman and Richards[19] and Gérard,[20] with ingenious technics, introduced colloidal and particulate matter into the proximal end of the proximal convoluted tubule. They found these substances accumulated in visible masses in the cells of the tubules, proving that reabsorption and alteration in dispersion could occur with other substances than glucose and hemoglobin.

In our laboratory, Dr. E. Canat has recently performed some very instructive experiments using trypan blue and T 1824 (Evans's blue), both of which are adsorbed onto albumin, the Evans's blue very strongly. When 5 mg. of the latter dye is injected intraperitoneally in normal rats, no dye appears in the urine, and the proximal convoluted tubules show none after six hours and only very faint staining even after twenty-four hours. The dye is in small vacuoles, barely visible in unstained frozen sections and invisible if the ordinary eosin staining has been used. However, if rats with severe proteinuria (100 to 400 mg. a day) are given similar injections, the urine becomes intensely blue. As much as 0.7 mg. of dye may be excreted the first day, varying with the severity of the proteinuria — roughly 1 mg. of dye per 500 mg. of protein in the urine. But the tubules in the animal's kidney (only a small stump of one kidney remains in the experimental rat) show large, intensely blue granules of dye, and large numbers of such granules may be seen within six hours. The proximal convoluted tubule contains the most dye when the loss in the urine is most intense, but even when no dye escapes in the urine small amounts gradually appear in the tubules. This is interpreted to mean that small amounts of serum protein, with adsorbed dye, are constantly escaping into the glomerular filtrate and being reabsorbed by the tubules. The dye is concentrated in vacuoles after being split off from the protein, and faintly visible amounts occur in the normal animal after many hours. When proteinuria is present, large amounts of dyed protein leak out of the glomeruli, the tubular cells reabsorb large amounts but much escapes in the urine, large granules of dye, split off the protein and concentrated by the tubule cell, become visible in the cytoplasm in a few hours. The analogy with hemoglobin and with glucose is very close, for with these substances what accumulates in the tubules is a polymer or a split product of what was present in excess in the glomerular filtrate. The tubule not only reabsorbs but also "works over" these substances.

With Drs. Canat and L. G. Woolley, I have used Winton's technic (perfusion of kidneys with ice-cold serum) to obtain large quantities of glomerular filtrate. That the glomerular membrane is not damaged in successful experiments can be verified by the observation that hemoglobin added to the perfusate appears in concentrations only 2.5 to 3.0 per cent as great in the glomerular filtrate. In rabbits, the protein content of glomerular filtrate, obtained in volumes as high as 3 cc. a minute, when the tubules are paralyzed by cold, varies from 15 to 22 mg. per 100 cc. The glomerular filtrate thus obtained is water-clear with no tinge of yellow; it is, as Bickford and Winton[21] showed, a pure ultrafiltrate with the glucose and chloride content of the plasma, and its protein content is about that of spinal fluid. Thus, the dye experiments receive confirmation regarding the probable presence of protein in the filtrate. It is hoped that this technic, applied to kidneys of animals with proteinuria and previously tested with hemoglobin, can give some information of the threshold level at which proteinuria occurs with a rise in protein content of glomerular filtrate.

It is well known that in the intense tubular damage due to severe hemoglobinuria or Bence-Jones proteinuria or with the crush syndrome (myoglobinuria), little or no fat appears in the tubules. In these cases, plasma lipids are normally low. In cases of Bright's disease with edema, with much fat in the tubules, plasma lipids are high, and this lipid is colloidal and often behaves as though adsorbed to globulin. The lipoid is high when the albumin is low, suggesting that when the body can no longer supply colloidal protein to maintain oncotic pressure of the blood, it begins to mobilize colloidal fats. In any event, the blood is presumably the source of the fat that appears in the tubule cells, as it is of the iron in hemoglobinuria, the glycogen in diabetes and the Evans's blue in rats with severe proteinuria. Degenerative tubular changes, as Ekehorn[7] and Govaerts[22, 23] suggested, are not the cause but the result of fat and protein accumulation in the tubules. With proteinuria, the cortex of the kidney is swollen, hyaline and granular masses occur in the tubules, and desquamation (up to 100,000,000 epithelial cells a day in the sediment) follows this over-engorgement with reabsorbed materials. Such desquamation is easily repaired, as evidenced by the mitoses seen in these cases, and the tubular functions are scarcely altered. Cats normally have far more fat[24] in their tubules than is seen in the severest human nephrosis, and of course they too have excellent tubular function. Until the tubules are severely altered, as in anuric cases of hemoglobin or myoglobinemia,[25] the degenerative change secondary to reabsorption leaves renal function almost intact. It is not impossible, however, that chronic proteinuria lowers the threshold at which protein leaks out of the body, and severe tubular changes may diminish capacity to reabsorb protein. In any event, the chief and the primary change is the rise in protein in the glomerular filtrate to levels at which tubular reabsorption no longer keeps pace with the supply. If the normal man has a threshold for spilling protein at 40 mg. per 100 cc., and normally has only 15 mg. per 100 cc. in the filtrate, he would have, in his 180 liters of filtrate a day, 27 gm. of protein, with none in the urine. At 41 mg., he would have 74 gm. in the filtrate and 2 gm. in the urine; if his threshold fell to 30 mg., with 41 in the filtrate, he would have 74 gm. in the filtrate and 20 gm. in the urine. These hypothetical figures give an idea of the way in which a slight rise in protein (or glucose or hemoglobin) in the filtrate can cause a huge increase in the urinary loss.

Proteinuria offers problems of diagnosis in medicine, pediatrics, surgery and obstetrics. Many types of investigation may be required to elucidate the cause in some cases, but no technic is more frequently of value than the quantitative study of the rate of loss of protein, cells and casts in the urine. Richard Bright set a bad example by sending his patients' blood and urine to Bostock, since in any field real skill is achieved only by those who thoroughly master and constantly employ the special technic necessary for study. It is as essential for the renal specialist to look at sediments obtained under standard conditions as for the heart specialist to do his own auscultation or the dermatologist to look at the entire skin surface.

There is evidence from the work in many laboratories on which to base an understanding of proteinuria and associated renal changes. This basis fits in with the knowledge of glycosuria and hemoglobinuria. But it leaves the central problem — the nature and cause of altered glomerular permeability — unanswered. More careful and constant scrutiny of patients and more ingenious and well-planned experiments will undoubtedly unravel much of this fundamental mystery.

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MEDICAL PROGRESS

REGIONAL ANESTHESIA

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THE current progress of regional anesthesia is characterized by an interest in its military application, by increasing confidence in spinal and fractional spinal anesthesia, and by widening the use of block technics in the therapeutic field. Studies of anoxia, respiratory drive and vascular collapse have helped to rationalize the conduct of both spinal and general anesthesia.

Lahey[1] has pointed out that trained anesthetists in military service will contribute definitely to a lowered surgical mortality rate. In their hands, local anesthetics may be differentiated on the basis of effect on wound healing,[2, 3] fractures may be remobilized,[4] and the use of spinal anesthesia will develop both in surgery and in the treatment of shock from injuries to the lower extremities.[5]

NEW AGENTS

Of the newer drugs available for regional anesthesia, monocaine hydrochloride,[6] an isomer of procaine, holds some promise. Somewhat more toxic than procaine, it is more stable to room temperature and light and equally stable to sterilization, has a distinctly greater affinity for motor nerves, and is clinically reported to afford more lasting anesthesia.[7]

Alkaline salts of procaine borate[8] are less irritating than the commonly used hydrochloride, but deteriorate rapidly at room conditions. Potassium salts of procaine afford more lasting anesthesia, but are immoderately irritating.

Laboratory studies have suggested that Intracain[9] might afford anesthesia twice as lasting as equivalent doses of procaine, but clinical trials have not confirmed this.

A group of piperidine derivatives[10] has been studied and listed as PT-1, PT-4, PT-6, PT-14 and PT-19. Of these, PT-19 appears most promising, providing spinal anesthesia twice as lasting as procaine. In infiltration anesthesia, it is more toxic and less efficient than procaine. Clinical trial has not been reported.

Neothesol in almond oil[11] has been used without complication in 427 cases to secure prolonged anesthesia in abdominal wounds by infiltration during surgery. Research work on cats, with placement of this oil in healing wounds, revealed on microscopy little or no resulting reaction.

Isoamyl cupreine[12] (eucupin in oil and in Ringer's solution) has been utilized to infiltrate and to block operative wounds of the upper abdomen. Postoperative vital capacity is increased, and sequent analgesia (sometimes without anesthesia) decreases the patient's discomfort. Laboratory work suggests that accidental intrapleural injection of local agents in oil during nerve-block procedures may have no lasting ill effect.

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Therapeutic Uses

The medical literature records an increasing use of local anesthesia in therapeusis. The antiseptic agent, rivanol,[2] combined with a local anesthetic, is suggested as an adjunct in the care of contaminated, painful wounds. This suggestion is timely, since local anesthetics derived from *p*-aminobenzoic acid (procaine) are antagonistic to the action of sulfonamide drugs when both are present in infected tissue.[3] Local anesthetics in oil for prolonged anesthesia, or repeated paravertebral block of abdominal wounds, may decrease postoperative pulmonary complications. Regulated cell reception of a local agent in a slowly absorbable, nontoxic, gelatinous medium is claimed to have a similar effect.[13]

Local infiltration facilitates early mobilization in the treatment of fractures of nonweight-bearing bones and minor injuries,[4] for example, those involving the back, shoulder, hand, wrist, ankle and foot.[14] Fractured ribs may require only one or two injections for complete rehabilitation.[15] Asthma and age are particular indications for such care. Anesthetic block is an adjunct to the treatment of fibrositis or bursitis,[16] and offers temporary relief as well as frequently prolonged remission of neuritides, particularly the trigeminal, occipital, cervical, brachial, intercostal or sciatic types.[17] Pain in amputation stumps may be interrupted or ended. Sympathalgia or causalgia of the face, traumatic "hard hand," or hyperhidrosis of the upper extremities may be improved by block of the stellate ganglion.

Articles by Bartlett[18] and Seldon[19] evaluate regional-nerve-block technics for surgery below the clavicles. Bilateral intercostal block is simple and serves admirably for plastic procedures on the upper abdominal wall, herniorrhaphy and closure of colostomy. Subtotal gastric resection in the patient who is a poor risk may be done readily under field block, with supplementary splanchnic block through the operative incision.[20, 21] By this method, Finsterer[22] reports good anesthesia in 90 per cent of 2442 cases; de Takats[23] reports success in 87 per cent of 1292 operations. General or fractional spinal anesthesia is needed for total gastric resection.

Tuohy,[24] of the Mayo Clinic, outlines excellent regional-block technics for saphenous ligation or femoral herniorrhaphy. He also describes procedures for caudal and transsacral blocks for anoperineal surgery and for work on the lower extremities.

Fetal and maternal risks are at a minimum under various types of caudal or, preferably, infiltration anesthesia.[25, 26] Results under the latter are consistent; perineal relaxation is notable, facilitating such procedures as low forceps, Scanzoni's maneuver, manual rotation, spontaneous breech and episiotomy. Anesthesia persists for one and a half to two hours.

The paravertebral sympathetic block for thrombophlebitis is an outstanding therapeutic application of regional anesthesia. All patients with superficial thrombophlebitis with pain and edema should have block and conservative measures.[27, 28] In patients with deep phlebitis without lung infarction, those under forty years of age need sympathetic block and conservative measures. In those from fifty to seventy years of age, 7 per cent suffer pulmonary infarcts, and ligation with block should be done early. In patients over seventy years of age, 20 per cent are likely to have pulmonary infarcts; in this group, ligation is obligatory, and blocks may be palliative.[29] Ten cubic centimeters of 1 per cent novocain with epinephrine — 1:200,000 at the first, second, third and fourth lumbar vertebras — will give relief in ten minutes and marked improvement in an hour. Blocks should be repeated one or more times daily until the temperature levels. Lumbar paravertebral block will improve claudication if sympathectomy is indicated.[30] If done preoperatively, this procedure prevents ischemic gangrene after surgery on large arteries.[31]

Paravertebral dorsal procaine block eases pain in postoperative upper abdominal wounds and raises the postoperative vital capacity, which is usually between 20 and 40 per cent of normal, to 90 per cent of normal. This permits hyperventilation and cough, with less tendency to atelectasis.[32]

Paravertebral alcohol injection may be preceded by diagnostic paravertebral cervicodorsal block for the relief of anginal pain or asthma.[33]

X-ray studies of 2 per cent novocain in opaque oil show that in epidural anesthesia in human beings, the onset of anesthesia follows diffusion of the agent out along the nerve roots.[34] This method may be used caudally for sciatica, dorsally for relief of intractable pain in carcinoma, at about the seventh cervical to the second dorsal vertebra for angina or asthma, and at selected levels for thoracic surgery. In the thoracic field, spinal anesthesia is less desirable than epidural anesthesia, which does not diffuse to bulbar centers; the effect can be limited to a few spinal segments, the blood pressure fall, nausea and vomiting are less, and "puncture headache" is decreased.[35] A mixture of pontocaine and procaine may afford anesthesia for two hours or longer.[36] Some danger results if an accidental puncture of the dura is made, in view of the large amounts of agent commonly used (20 to 50 cc. of 2 per cent novocain).[37]

Spinal Anesthesia

Of the agents available for spinal anesthesia, procaine continues to be used most commonly; however, there is a tendency toward the use of solutions weighted with 6 to 10 per cent glucose to facilitate ready gravitation of the agent to desired sensory spinal levels.[38] In Britain, Percaine (nupercaine),[39] 1:200 with 6 per cent glucose, as well as pontocaine-glucose, is utilized; in this country, pontocaine hydrochloride, 1 per cent in 10 per cent glucose, is gaining popularity and has been used at the Lahey Clinic in about 8000 cases without a serious complication referable to the agent.

A significant contribution to subdiaphragmatic surgery during the last decade is the development of fractional (continuous) spinal anesthesia. This type, maintained with rubber tubing and an indwelling soft German-silver spinal needle, has now been used in a sufficient number of cases to permit evaluation.[40–42] It is particularly adapted to prolonged surgery in patients who are poor risks, in whom relaxation is desirable. The incidence of complications referable to the anesthesia is not greater than with any other comparable method.

Spinal anesthesia has been carried to cervical levels with some success, permitting such operations as mastoidectomy. On physiologic bases, this cannot be approved for it brings the agent perilously close to medullary centers, and depends on the selective paralysis of the sensory to the motor (phrenic) nerves.[43]

Bourne,[44] who has modified the Etherington-Wilson technic for nupercaine to secure satisfactory spinal anesthesia for thoracic surgery, believes that with the use of this agent breathing is quieter, paradoxical breathing is decreased and secretions are less, and that the prolonged postoperative anesthesia aids convalescence. In these cases, intercostal or epidural block seems a more rational substitute for general anesthesia. Intratracheal cyclopropane or ether may be wholly preferable.

In the past, the fall in blood pressure experienced with spinal anesthesia was a barrier to its use for cesarean section. However, Heard[45] regards this disadvantage of spinal anesthesia as slight when the depressing effect of ether on the mother and fetus is considered. This bad effect of ether reaches its peak in abnormal fetuses or in heavy sedation of the mother. Circulatory, respiratory and genitourinary complications are reduced when spinal anesthesia is used, and the baby is not narcotized.[46]

Hunt and Lundy[47] favor local or field block for cesarean section. To this technic, they have added the intravenous administration of Pentothal Sodium when the uterus is opened because at that point some of their patients complained of distress. Oxygen or 50 per cent nitrous oxide and oxygen may be given for a few minutes if much Pentothal seems indicated.

Caudal Anesthesia

Caudal anesthesia may also be utilized in parturition. Lahmann and Mietus[48] report 400 cases without incident, except a 2.5 per cent failure to produce anesthesia. Perineal relaxation occurs, contractions are painless, the uncontrolled "push urge" is diminished, separation and involution are normal, and the mother is rested. Continuous caudal anesthesia is also being used in obstetrics.[49]

Supplementary Anesthesia

Premedication should be considered supplementary anesthesia, and is a vital factor in spinal anesthesia of any type. Pantopon in adequate doses, used instead of morphine, tends to decrease the incidence of nausea and vomiting.[50] Scopolamine is preferable to atropine, creating twice the drying effect and twice the psychic depression. The depressant effect of morphine, too, is more satisfactorily counteracted.[51] When the drug is combined with Pantopon and a barbiturate if indicated, the exciting effect of scopolamine largely disappears.

Extensive surgery under spinal anesthesia may necessitate additional supplementary anesthesia, particularly if mesenteric traction initiates nausea and vomiting. The high concentration of oxygen that may be administered with cyclopropane is desirable but, in intra-abdominal surgery, may be contraindicated by the use of the cautery. In these cases, Pentothal Sodium administered by fractional doses into a continuous intravenous-drip apparatus is convenient. However, its depressing effect on the respiratory drive mechanism[52] makes logical the simultaneous administration of nitrous oxide and oxygen, to prevent the patient from going into central respiratory failure.[53]

Supportive Measures

The value of an adequate oxygen supply to patients under prolonged spinal anesthesia with associated low blood pressure cannot be overemphasized. Arterial oxygen saturation, 5 to 45 per cent below normal in the head, can cause cerebral damage and a marked increase in water content (edema) of cerebral tissue.[54] This depression, added to the accepted histotoxic depression of Pentothal or preoperative barbiturates, helps to explain many of the accidents that have retarded the progress of spinal anesthesia.[55]

The dangers of anoxia are inseparable from the problems of shock. Certain tests may detect shock during its earlier stages when significant changes are noted in the hematocrit, the specific gravities of the plasma and whole blood, and the plasma protein level.[56] The primary fall in blood pressure in spinal anesthesia probably depends on peripheral vasomotor paralysis. Other mechanisms accounting for the fall in blood pressure beyond the level caused by peripheral vasodilatation, suggested by Nowak and Downing,[57] are the central effects of vasomotor change and anoxia, in addition to medullary paralysis caused by direct extension and vascular absorption of the drug. When these tests or clinical observation indicate that shock is developing, electrolytes may be restored with physiologic saline, capillary tone with vasopressors and cortical extract, and the circulation sustained with blood or plasma.[58, 59] Meanwhile, oxygen should be available, flowing in concentrations of about 60 to 75 per cent. Carbon dioxide should not be administered to spinally anesthetized animals or patients, since it lowers their blood pressure,[60] has little or no effect on respiration and contributes to already present anoxia.[61]

To combat the fall of blood pressure common with spinal anesthesia, ephedrine has proved to be the vasopressor of choice. As a working hypothesis, one may assume that spinal anesthesia blocks the sympathetics to the adrenals and lowers the epinephrine available. Then, if ephedrine is administered, it blocks amine oxidase, the epinephrine neutralizer, freeing "frozen" epinephrine. If ephedrine and fluids do not raise blood pressure and speed circulation, a small intravenous dose of epinephrine helps the depleted stores.[62] A mixture of Pitressin and ephedrine elicits a more rapid and prolonged effect than ephedrine alone.[63] Too liberal administration of these pressor drugs may be somewhat dangerous in patients with arteriosclerosis or coronary disease. Epinephrine alone is efficacious, but its effect curve is too short and peaked.[64]

An intermediate substitute for epinephrine is Synephrin tartrate.[65] A dose of 400 mg. secures a marked systolic rise, a slight diastolic rise and a slight fall in pulse rate. The onset is rapid, and the duration is over one hour.

Gaining favor is the related Neosynephrin, longer acting than ephedrine, and having less pressor and cardioirritant effect and more sympathetic side-effects.[66] The associated apnea sometimes observed is probably a carotid and aortic-body response to rapid elevation of blood pressure. The threshold doses are 2 mg. subcutaneously and 0.4 mg. intravenously; the average effective doses are 5 mg. subcutaneously and 0.8 mg. intravenously. Overdosage creates cardiac irregularity and electrocardiographic variations.[67]

Adrenocortical hormone (cortin) and desoxycorticosterone have been suggested as aids in the maintenance of blood pressure and in the treatment of shock. Desoxycorticosterone has no established value here. However, cortin has at least three uses: it protects against traumatic and adrenalin shock[68]; under anoxic conditions, it is needed to mobilize catabolic protein and spare liver glycogen[69]; and in the treatment of shock, it has a definite use in mobilization of fluids, particularly parenteral saline and plasma.[70]

Complications

The complications arising under regional anesthesia are so varied as to prohibit an integrated discussion, but certain facts stand forth. Lyford[71] reports that patients with normal respiratory systems before surgery have postoperative respiratory infections in about 6 per cent of cases; there is no relation to type of anesthesia. He states that patients with low-grade respiratory infection before surgery will have acute postoperative episodes two and a half times more frequently with spinal anesthesia than with inhalation anesthesia (spinal 39.5 per cent; ether 13.5 per cent; cyclopropane 17.5 per cent).[72] These figures are not in accord with the very excellent work of King[73] at the Massachusetts General Hospital.

Postoperative atelectasis — much of the "ether pneumonia" of other days — has yielded well to catheter aspiration with conservative hyperventilation, together with limitation of sedatives. If this fails, therapeutic bronchoscopy is indicated, even in very sick patients, for pneumonitis in a collapsed lung is far more inimical than bronchoscopy performed by fairly skilled persons.[74, 75]

The prevention of atelectasis may be possible. Capelle[76] observed that the usual postoperative vital capacities of 20 to 40 per cent of normal were increased to 90 per cent of normal by continuous local anesthesia of abdominal wounds. Gius,[32] impressed with Capelle's work, tried paravertebral intercostal block for atelectasis occurring after appendectomy in children, with notable success. Subsequent studies of vital capacities after abdominal surgery indicate that respiratory excursion, cough and movement are markedly facilitated by prolonged or repeated block anesthesia. Infiltrating such a wound at the end of an operation sometimes affords later analgesia without pinprick anesthesia.[12] Starr and Gilman[77] report 23 per cent increase in postoperative vital capacity with blockage of the sixth to the tenth dorsal nerve inclusive.

The selection of cases (preoperative respiratory conditions) and of operations (nephrectomy and

cholecystectomy) for therapeutic intercostal block may markedly decrease the incidence of atelectasis. Few patients will object, for marked comfort is afforded.

The hypnotic drugs, such as Pentothal and Evipal, may create surgical or postoperative depression. At a deep anesthetic level (stage III, plane II), the cerebral respiratory centers may cease to function, and respiration becomes dependent on anoxic stimulation of the carotid sinus and aortic body. Carbon dioxide may pile up in the circulation until it actually interferes with oxygen transfer to tissues.[61] At this point, a sudden supply of oxygen by mask may throw a patient into irreversible apnea.[52] Such hypoxia is not well tolerated by patients under spinal anesthesia; therefore, hypnotics as supplemental anesthetics must be handled carefully. Postoperative hypnotic depression may be counteracted by benzedrine.[78] As yet, no relation between hypnotics and the toxicity of sulfonamides has been established.[79]

Convulsions after bronchoscopy under topical pontocaine hydrochloride have been controlled by three 2-cc. doses of Dial.[80] Research suggests that in such cases calcium levulinate, calcium gluconate and calcium chloride are protective, in that order.[81] Calcium chloride and Salyrgan protect against procaine convulsions, but only if injected with the anesthetic.[82] Barbiturates given preoperatively protect, but increase the danger if injected with the anesthetic. Barbiturates used to excess for the control of cocaine convulsions cannot be counteracted as usual with Metrazol or picrotoxin; patients may have convulsions without awaking.[83]

Further complications described in the literature are abducens paralysis, meningitis,[84] ventricular fibrillation, urinary retention and cauda-equina injury. Urinary retention, occurring in about 3 per cent of cases, usually responds to constant or tidal drainage with sulfonamide irrigations. Resection of a portion of the bladder sphincter has cured a refractory case.[85] Reported cauda-equina injury was seen in several patients given Spinocaine. Ferguson and Watkins[86] reported 14 such cases among seventeen hundred spinal anesthesias under durocaine, a procaine-gliadin-glycerine solution in 15 per cent ethyl alcohol.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

CASE 28431

PRESENTATION OF CASE

First admission. A fifty-eight-year-old man was admitted to the hospital because of a feeling of substernal oppression.

One year prior to admission the patient began to suffer with attacks of substernal oppression and chest tightness after exertion, without severe pain. These attacks were reduced in severity by rest and lessened activity. During the week prior to admission he suffered with almost daily attacks of "fluttering and bumping of the heart," each lasting about five minutes. During the attacks he had a feeling of impending danger; however, no remarkable pain developed.

The family history was noncontributory. Twenty-nine years previously the patient had had malaria, but since then he had had no chills. He did not recall having had any other tropical diseases.

Physical examination revealed a well-developed and well-nourished man in no apparent distress. The chest was of an emphysematous type but was otherwise normal. The heart when outlined by percussion was questionably slightly enlarged. The sounds were distant and faint. A blowing systolic murmur was audible at the apex. The abdominal examination was unremarkable, and no masses were felt.

The blood pressure was 120 systolic, 80 diastolic. The temperature was 99°F., the pulse 90, and the respirations 20. Several hours after admission the pulse was found to be 49, and during hospitalization it ranged between 50 and 60.

Examination of the blood revealed a red-cell count of 4,730,000 with a hemoglobin of 85 per cent, and a white-cell count of 10,800 with 68 per cent polymorphonuclears, 26 per cent lymphocytes and 6 per cent monocytes. The urine was normal except for a rare red cell and 2 white cells per high-power field in the sediment. The blood Hinton test was negative. The blood nonprotein nitrogen was 23 mg. per 100 cc. The sedimentation rate was reported 1 mm. in fifteen minutes, 4 mm. in thirty minutes, 6 mm. in forty-five minutes and 8 mm. in sixty minutes. An electrocardiogram demonstrated a normal sinus rhythm of 60 per minute. The P-R interval was 0.19 second; T_3 was inverted, and there was a slight amount of left-axis deviation. The patient was discharged after three days and advised to take Nembutal at bedtime and 3 gr. of quinidine sulfate fifteen minutes before each meal.

Second admission (nine months later). The patient was readmitted because of a recurring fever of about 101°F., weakness and night sweats. These symptoms developed three months after discharge and seemed similar to the fever and chills with which he had suffered in the attack of malaria. A typical episode began at night with chills and trembling, followed by fever of 103°F., vomiting and sweating, which gradually decreased during the night leaving him weak but quite all right during the next day. Six weeks before entry he again began to have night sweats and evening fever, and these symptoms continued intermittently to the day of admission, being slightly mitigated by sulfadiazine administered by his local physician. Three weeks before entry his face became swollen and pink, and this skin flush gradually extended over the chest and abdomen and lasted several hours.

Physical examination was similar to that noted above except that the patient was obviously feverish. The soft systolic murmur was audible over the entire precordium, but best heard at the apex, and was transmitted into the left axilla.

The blood pressure was 110 systolic, 60 diastolic. The temperature was 101°F., the pulse 85, and the respirations 24.

Examination of the blood revealed a hemoglobin of 16.1 gm. and a white-cell count of 8000 with 75 per cent polymorphonuclears, 17 per cent lymphocytes, 7 per cent monocytes and 1 per cent blasts. The red blood cells appeared fairly normal; the platelets were slightly increased. No malaria parasites were seen. The urine examination was similar to that of the previous admission. A stool examination was negative for mucus and blood. Repeated examinations of the blood were not remarkably different, and no malaria parasites were found. Agglutination tests for undulant fever were negative, and the Widal test was negative. Repeated blood cultures revealed no growth.

An x-ray film of the chest demonstrated a small, rather sharply defined area of density in the right second interspace. There were areas of calcification in the right lower lung field, and linear scars in the left costophrenic angle. The heart was prominent in the region of the left ventricle. The aorta was tortuous. An electrocardiogram was similar to that taken during the previous admission.

During the nine days in the hospital, daily periods of fever up to 103°F. and sweating occurred in the evening, in spite of a brief course

of quinine sulfate. He was discharged with plans for readmission a week later. His fever had subsided somewhat.

Third admission (two weeks later). Since discharge he had had a gradual return of pyrexia, although for the first week at home his temperature was subnormal.

The physical examination had not changed except that the patient was definitely sicker.

The temperature was 101.4°F., the pulse 89, and the respirations 20.

The hemoglobin level was 10.3 gm., and the white-cell count 9200 with 64 per cent polymorphonuclears, 17 per cent lymphocytes and 19 per cent monocytes. No malaria parasites were seen in the blood smear. The urine examination was unchanged except for a + test for albumin. An electrocardiogram did not demonstrate any remarkable change. An x-ray film of the chest showed some hazy density extending upward and posteriorly from the right hilus, apparently in the apex of the right lower lobe.

The patient was discharged after six days without much improvement.

Final admission (two weeks later). Since discharge, he had had a subnormal temperature for one week and then it gradually rose from an evening peak of 99°F. on the first day of the week to 104°F. on admission, with profuse night sweats and daily remissions. He had been taking 40 to 80 gr. of aspirin each day and had recently developed some deafness and ringing in the ears. Three or four days before entry he noted the onset of attacks of rapid pulse, which often rose from 78 to more than 150. These changes occurred suddenly, lasted a few minutes and stopped suddenly. He had had twenty to twenty-five attacks during the four days prior to admission. They were accompanied by a burning headache and perspiration. All these symptoms ceased with cessation of the rapid pulse rate. There was no dyspnea or precordial distress with the attacks.

Physical examination revealed a flushed, apprehensive man with a hot, dry skin, who looked chronically, but not acutely, ill. The examination of the lungs was normal, except for equivocal dullness at both bases posteriorly and a suggestion of bronchovesicular breathing in these areas. A few fine rales were audible at both bases. The heart was markedly enlarged to the left in the anterior axillary line, and the apex impulse was felt in the left fourth interspace in the anterior axillary line. There was a moderately loud blowing systolic murmur over the entire precordium which was best heard at the apex and was transmitted to the left axilla. A somewhat rasping but soft systolic murmur was audible at the base and in the neck vessels. Along the left border of the sternum there was a midsystolic scratching sound, which seemed more like a precordial rub than transmission of a systolic murmur.

The blood pressure was 134 systolic, 74 diastolic. The temperature was 104°F., the pulse 90, and the respirations 32.

Examination of the blood revealed a hemoglobin of 9.7 gm. and a white-cell count of 5600 with 86 per cent polymorphonuclears, 10 per cent lymphocytes, 2 per cent monocytes and 2 per cent myeloblasts. The urine was unchanged, as was an x-ray film of the chest. In the flat plate of the abdomen the kidney shadows were obscured by overlying bowel but were not grossly abnormal. There was a small area of density overlying the right side of the sacrum. Further blood cultures revealed either no growth or obvious contaminants. The urine culture was negative. A stool examination was negative for blood, mucus, amebae and amebic cysts. An intravenous pyelogram was negative. A cephalin flocculation test was +++ at 24° C. and ++++ at 48° C., but in the liver-function test there was less than 5 per cent of the dye in the serum. One month after admission, tiny lymph nodes about 5 mm. in diameter, were felt in the neck on each side.

Since no diagnosis was established, the patient was empirically treated with a course of sulfadiazine by mouth, totaling 69 gm. in seventeen days. The blood level established apparently did not exceed 5.9 mg. per 100 cc. There was only a moderate reduction in the temperature during the course of sulfadiazine, and the patient was not remarkably improved. His fever continued to spike to 103 or 104°F. almost every day. Short courses of quinine sulfate and Atabrine were administered without obvious improvement. In spite of supportive and symptomatic treatment, the patient gradually became weaker. In the last week before death, three small lymph nodes became palpable for the first time in the groins, and the patient gradually became moderately jaundiced. Symptoms of fever and chills continued, and the patient died three months after admission.

Differential Diagnosis

Dr. Francis R. Dieuaide: In reading through the abstract, we note that the first complaints were those of anginoid attacks with runs of premature beats, that is, short attacks of paroxysmal tachycardia or possibly, though less likely, paroxysmal auricular fibrillation. These were repeated during the fourth admission. The mid-diastolic scratching sound along the left border of the sternum leads me to believe that the patient had a mild pericarditis at the time of the fourth admis-

sion. The blood pressure showed changes up and down, which were not, however, very great.

The blood changes include the finding of 19 per cent monocytes and 1 per cent myeloblasts on different occasions. It is not safe to attribute great importance to these. By the time of the last admission there was moderately severe anemia. The whole course of the patient, if we consider the beginning to be when he first had substernal discomfort, took about two years. The chills and fever lasted about ten months. These recurrent attacks of fever are very striking and must contain the central clue to the diagnosis. Apart from them, the findings point in three directions.

Certain findings relate to the heart. We know that the patient had substernal discomfort. He had paroxysmal attacks of tachycardia, with some variation in blood pressure, and developed marked enlargement of the heart to the left, with a loud systolic murmur. He apparently never developed congestive failure, had no sign of valvular disease and, during observation, had no hypertension. He probably had a short episode of acute pericarditis during the last admission. There is no reason to think he had a huge pericardial effusion as a possible explanation of the apparent enlargement of the heart.

Another direction in which our attention is drawn is the liver, since we are told that the patient had jaundice.

The third direction we must consider is suggested by the lymph glands in the neck and in the groin, which were small and apparently did not attract much attention. There were also some findings in the chest.

May we see the chest films, please?

DR. GEORGE W. HOLMES: The shadows spoken of in the chest are these small spots of calcification. I should not say that they had any particular bearing on this patient's story.

There is a moderate amount of tortuosity of the aorta, and the heart seems to extend a little farther to the left than usual, but in view of these other things I doubt if it is actually enlarged. The left ventricle is perhaps a bit prominent. The other things that were mentioned I think are of no importance. These lines are probably blood vessels; there is nothing characteristic about them.

DR. DIEUAIDE: Do you think anything of that shadow at the right hilus?

DR. HOLMES: No; one might say the pulmonary vessels are a little prominent. I do not believe that would be of any aid in the diagnosis or that one was justified in saying it was definitely abnormal.

DR. DIEUAIDE: I am very humble about this, but is that not a well-defined outline at the hilus — more so than usual?

DR. HOLMES: I do not believe so. I should not call it abnormal, much as I should like to agree with you. There is one strange thing about this. At one time his heart was said to have been enormously enlarged. I do not see any evidence of it in any of the films. If he did have enlargement, it apparently disappeared.

One would like to try to find out something about the size of the liver though these films were not taken for that purpose. It is not enlarged; if anything, it is small. Also you can see the outline of the spleen, and that does not seem enlarged. The kidney shadows are large as we see them on the film but no larger than a heavy man should have. They are magnified a good deal.

I am afraid most of my evidence is negative.

DR. DIEUAIDE: If we depart for a moment from this point and think about the febrile course, which is the most striking aspect of the case, we must run over a number of possibilities. In the first place there are many varieties of low-grade septicemia, including bacterial endocarditis, brucellosis and many other infections. This patient had a normal white-cell count, which is against many of these infections, but is not exclusive. He had no enlargement of the spleen, so far as we know, and no embolic phenomena (petechiae, subcutaneous nodules, nephritis, and cerebral or pulmonary embolism). He had no valvular disease of the heart, which puts out bacterial endocarditis. He had many negative blood cultures, which in the end one has to take as being against septicemia.

One should always consider tuberculosis. This man was fifty-eight years old; it is the fashion to think that people as old as that do not get tuberculosis and die of it, but this is a mistake — they do. On the other hand, in ten months, tuberculosis should declare itself in the chest, abdomen or meninges, unless one is dealing with some peculiar localization, such as the adrenal glands, of which there is no indication. Tuberculosis is not out of the question, but I do not believe it is the diagnosis.

Typhoid fever is always mentioned in these cases. Again, in ten months it should be picked up, and I think we can throw out the possibility.

Is this malaria after twenty-nine years, without any remissions after quinine, without any enlargement of the spleen and with death? No, I do not think the patient had malaria.

A new growth, such as lymphoma, has to be one of the possibilities that we think of seriously. Such a patient would be studied thoroughly for some localization of the tumor, and I assume that this was done. Could these little lymph nodes be the only signs of new growth? Could this shadow

in the chest be a mass of lymph nodes and be more significant than hilar vascular changes? Certainly this large group of possibilities cannot be left out.

Next, we must consider hepatic disease. The patient undoubtedly had some form of liver disturbance, since he had progressive jaundice. The abstract gives only the results of liver-function tests and does not mention palpability of the organ. Perhaps we may assume that it was not palpable. The flocculation test was positive, but the bromsulfalein test gave normal results. It might be of interest to know whether there was a long interval between the bromsulfalein test and death.

DR. TRACY B. MALLORY: There was a three-month interval.

DR. DIEUAIDE: In other words, the bromsulfalein test was done at the beginning of the third admission, and therefore it does not do what we should like it to do. If the patient had diffuse liver disease at the time of death, I am sure that the bromsulfalein test would not have been normal at the end. With serious disease of the liver as the fundamental cause of death, we should expect a declaration of the disease in a well-observed patient in the form of jaundice, ascites, liver enlargement, pain about the liver and evidence of hepatic insufficiency. Of these only jaundice appears. I do not believe that hepatic disease was the cause of death.

Phlebitis might be mentioned, but there is no lead to that — no localizing signs and no evidence of embolism.

Then, there are certain obscure diseases that have to be brought up. I suppose there are many others, but there are three which especially come to mind: disseminated lupus, sarcoid and periarteritis nodosa. So far as disseminated lupus and sarcoid are concerned, we have not the vaguest clinical suggestion that either of these conditions would have been found post mortem. Periarteritis nodosa could be the diagnosis, although there is a question whether patients have been seen with such fever as this. I hope someone else will speak about this point before we know the diagnosis. So far as I know, the febrile episodes are out of line with this possibility, but many of the other findings are not. In the first place, some of the cardiac events would fit in — the anginoid syndrome, with the substernal oppression, the variation in the blood pressure and even cardiac enlargement and pericarditis; the age and duration of the disease are compatible. The patient did not have the outstanding characteristic features of periarteritis, such as leukocytosis, eosinophilia, abdominal pain, nodules, enlargement of the spleen and so forth. However, all these findings are seen in only a very moderate percentage of patients with periarteritis nodosa.

As Dr. Mallory anticipated, I am unable to make a definite diagnosis. I am going to make periarteritis nodosa my first choice and put down neoplasm, perhaps lymphoma, in the next line.

CLINICAL DIAGNOSIS

Hodgkin's disease?

DR. DIEUAIDE'S DIAGNOSIS

Periarteritis nodosa?
New growth (possibly lymphoma)?

ANATOMICAL DIAGNOSES

Malignant lymphoma, Hodgkin's sarcoma type, involving lymph nodes, spleen and bone marrow.
Icterus.
Central necrosis of liver.
Atherosclerosis moderate: aortic, coronary and pulmonary.
Emphysema, moderate.
Cholelithiasis.

PATHOLOGICAL DISCUSSION

DR. MALLORY: This was obviously a most obscure case. At autopsy, we found several very slightly enlarged lymph nodes, a perfectly normal liver and a slightly enlarged spleen without any characteristic markings or nodules. It seemed to me impossible to make any diagnosis on the gross findings. When the microscopic sections came through, it was obvious that these not very important looking nodes all showed typical Hodgkin's disease, that there were many scattered Sternberg cells in the unimpressive spleen and that the major Hodgkin's disease was in the bone marrow, which was almost 75 per cent replaced by the disease. The heart was normal in size, and there was only 80 cc. of pericardial fluid. There was nothing to indicate that there had ever been a pericarditis. There was moderate emphysema.

A PHYSICIAN: Was there a mediastinal mass?

DR. MALLORY: There was very slight enlargement of the hilar glands, but no mass.

DR. DIEUAIDE: Did they show Hodgkin's disease?

DR. MALLORY: Yes.

CASE 28432

PRESENTATION OF CASE

A thirty-two-year-old man was referred to the hospital because of fever, chills, diarrhea and jaundice.

Three weeks prior to admission he noticed extreme weakness on arising in the morning. He went to work but was forced to return home in the afternoon because of extreme weakness and fatigue. Because he did not feel up to par, he took a laxative, which produced watery stools for two days. At no time was blood noticed in the stools. He was seen on the following day by his physician, who told him that his temperature was 102°F. He was urged to enter a hospital, but he postponed this for four days, during which period he had several chills and suffered with vague poorly defined abdominal pains largely confined to the epigastrium but not well localized.

On admission to a community hospital he was very dehydrated and prostrated, obviously quite ill. He was treated with sulfathiazole, apparently by mouth and, after 45 gr. of the drug had been given, was found to be jaundiced. For this reason the drug was discontinued. Laboratory studies revealed a red-cell count of 4,200,000 with 80 per cent hemoglobin, and a white-cell count of 6000 with 90 per cent polymorphonuclears. Two blood cultures were reported negative. A test for Weil's disease done at a federal laboratory was reported negative. Repeated Widal tests were negative. The urine was negative except for a rare red blood cell, an occasional granular cast and many bacteria. The stool examination revealed only a ++ guaiac test. The stools did not contain gross blood and were not tarry. The van den Bergh test and icteric index were first reported 10.7 mg. per 100 cc. and 13, respectively, and when repeated several days later, were 5.27 mg., and 6.7. During the two weeks in the hospital he became progressively anemic and the white-cell count rose to 12,000. He became progressively worse, the illness being characterized by weakness, chills and evening temperature elevations to 106.8°F., with a morning temperature of 100°F. At times he complained of a slight pain localized in the upper abdomen. Bowel movements were regular, but the stools seemed to be lighter in color than normal. There were no urinary symptoms. Because no diagnosis could be established, he was transferred to another hospital for further study. Here the picture remained essentially as previously noted except that a blood culture demonstrated gram-positive cocci in short chains and many diplococci. When cultures were repeated, no cocci were found but many large gram-positive (*sic*) rods were cultured, which proved to be Friedländer's bacilli. X-ray films of the abdomen demonstrated a dense somewhat homogeneous soft-tissue mass, which filled the entire epigastrium. There was an irregular fleck of calcium to the left of the third lumbar interspace, which was thought to have no connection with the soft-tissue shadow. The liver and spleen appeared moderately enlarged. The shadows of the kidneys and psoas muscles were distinctly seen. The gas-filled transverse colon was depressed and lay just above the pelvis. Re-examination of the abdomen with a swallow of barium outlined the stomach, which was flattened and displaced to the left and downward by the previously noted round soft-tissue mass. There was no obstruction. There was no definite evidence of intrinsic disease of the stomach, and the duodenal loop was small. The patient was started on sulfadiazine, and after eighteen hours, the blood level was 7.1 mg. per 100 cc. He had three chills during the two days' stay at the hospital, and the temperature fluctuated between 100 and 104°F. He had no specific complaints other than weakness, chills and fever. No diagnosis was established. The patient was brought to this hospital for further study and care.

The family history was not contributory. The patient lived in the country and was accustomed to drinking well water, which he obtained from his own well. This was said to be 18 feet deep, and the water was declared good. He used a privy. He stated that he had always used raw milk until three weeks prior to the present illness, when he changed to condensed milk since raw milk was difficult to obtain. He had never been immunized against typhoid fever and had been unsuccessfully vaccinated against smallpox. He smoked occasionally and did not use alcohol excessively. Ten years previously, when admitted to a government hospital for a circumcision, a scar was found on the glans penis. He denied having had a penile sore or having had gonorrhea. Serologic tests at that time were negative for syphilis.

Physical examination revealed a feverish, jaundiced young man perspiring profusely and obviously acutely ill. Examination of the lungs and heart was negative, except for a soft apical systolic murmur. The abdomen was moderately distended and tympanitic. No masses were palpable, and the liver edge could not be percussed because of abdominal tympany. The upper border of liver dullness seemed elevated, and the area of splenic dullness was increased. There was slight tenderness in the right upper and right lower quadrants. Normal peristaltic sounds were audible. The rectal examination was negative.

The blood pressure was 125 systolic, 84 diastolic. The temperature was 102.4°F., the pulse 99, and the respirations 24.

The examination of the blood revealed a red-cell count of 2,410,000 with a hemoglobin of 50 per cent and a hematocrit of 25 per cent, and a white-cell count of 17,100 with 91 per cent polymorpho-

nuclears. The urine was acid in reaction, had a specific gravity of 1.018 and showed a + test for albumin. The van den Bergh was 2.5 mg. per 100 cc., and the chloride 96.9 milliequiv. per liter. The prothrombin time was 25 seconds (normal, 20 seconds), the protein 6.1 gm., the blood sugar 94 mg. and the nonprotein nitrogen 23 mg. per 100 cc., and the bicarbonate 20.0 milliequiv. per liter. A flat plate of the abdomen demonstrated barium scattered throughout the colon, with a portion of it in the rectum. The psoas shadows were slightly indistinct. The kidneys were faintly seen. There was increased haziness throughout the upper abdomen, but no definite mass was seen. The splenic flexion of the colon was definitely lower than normal.

A course of sulfadiazine was started, and a transfusion of citrated blood was administered. The second day after admission, the patient was operated on.

Differential Diagnosis

Dr. Allen G. Brailey: In striving to unravel this story, we might begin by asking what was the nature of the disease that made this patient ill, and it seems obvious that it was acute infection. Then we might ask in what organ or system the infection had a primary focus, and there again it seems obvious that it had to do with the biliary tree. An illness that is characterized by extreme weakness, chills, fever and the development of jaundice and anemia might well be due to pylephlebitis. The commonest source of such a pylephlebitis is the appendix. The organisms enter the portal vein and cause septic thrombi and then emboli, which lodge here and there throughout the liver and result in the production of multiple abscesses. Appendicitis may occasionally produce very few symptoms, but in this case there is no hint whatever of antecedent appendiceal infection unless one wants to attach importance to the fact that there was tenderness in the right lower quadrant. I feel hesitant about making a diagnosis of appendicitis on such slender evidence as that. Of course, pylephlebitis might rarely originate from infection elsewhere in the gastrointestinal tract. There is the note of a positive guaiac test from one hospital, so that a certain amount of occult blood had been present. I should think there were adequate reasons for that amount of blood in an inflammatory process in the biliary tree and areas surrounding the duodenum, but I suppose he might have had a small ulcer somewhere in the gut, which initiated a pylephlebitis.

Then, there is acute suppurative cholangitis, which is an interesting disease and is sometimes secondary to infection elsewhere, such as pneumonia, influenza or, occasionally, typhoid fever. Most commonly it follows cholecystitis. When it originates primarily in the biliary tree, it usually follows obstruction of the biliary tract, such as that from stone, tumor or echinococcal cyst or, occasionally, from intestinal worms, which may obstruct the common duct. When it is due to biliary obstruction one expects to find the biliary tree widely dilated and filled with pus. The walls are thickened, there are multiple small periportal abscesses, and the history is quite like this, with extreme weakness, a spiking septic temperature, the gradual development of jaundice and, usually, a fairly rapid fatal termination. No obstruction to the biliary tree is necessary, however; it may develop as a result of a blood-borne infection originating elsewhere or, sometimes, de novo. The organisms involved are usually streptococcus, *Staphylococcus aureus*, pneumococcus and others. I am somewhat dismayed by the bacteriologic situation. Four blood cultures were taken. Two were negative. One showed gram-positive cocci in short chains and diplococci, which I should guess were contaminants. Then one showed gram-positive bacilli that were said to be Friedländer's bacilli. This bacillus is a gram-negative, encapsulated organism, also known as *Bacillus mucosus capsulatus*, and is responsible for 5 to 7 per cent of pneumonias, which are highly fatal and characterized, among other things, by sticky tenacious sputum. It is also an interesting organism in that it is closely related to the colon group and can at times be isolated from intestinal contents. It may give rise to infection of the intestinal tract and of associated organs. I do not know that any great importance need be attached to this report of Friedländer's bacillus; but still, if it was found, it probably indicates a bacteremia. Perhaps the infectious process was due to that organism.

Now let us consider this epigastric mass of which we have this account that is so indefinite. It might, of course, have been a tumor, a lymphoma or another mass, that served as the cause of the obstruction of the biliary tract. It might also have been an extrahepatic abscess. Abscesses within the liver not infrequently, or at least occasionally, rupture through the capsule and produce such a mass. The associated infection of the common duct and gall bladder might have done likewise. Of course, if there were such an extrahepatic abscess that developed slowly enough it would have been walled off by the omentum and localized to the upper abdomen.

I do not know what this man had. On the whole, I think he had an acute suppurative cholangitis; whether this was initiated by pylephlebitis I do not know. I do not believe the surgeons knew

either. I think he was probably operated on with the idea of draining the obvious septic focus, either in the biliary tree or an extrahepatic abscess.

DR. RICHARD H. SWEET: Would it be fair to make a comment from a clinical standpoint on the description of the mass?

DR. TRACY B. MALLORY: Yes.

DR. SWEET: There was a mass. I thought that it was liver. It felt like a mass within the liver in the epigastric region and was not tender.

DR. BRAILEY: Then it might have been an intrahepatic abscess that deformed the normal liver margin. I doubt if an abscess of the liver would of itself be of sufficient size ordinarily to be palpable in the epigastrium. Do you think it might have?

DR. SWEET: Yes, it might.

DR. BRAILEY: Then perhaps the mass was all inside the liver.

DR. MALLORY: Then your preference is cholangitis with multiple abscesses in the liver?

DR. BRAILEY: I will stick to that.

DR. MALLORY: Does anyone else care to express a preference?

DR. REED HARWOOD: I think the possibility of Weil's disease should be mentioned in the list of diagnoses.

DR. MALLORY: That was thought of and serum for an agglutination test was sent to Washington; the report was negative.

DR. SWEET: If my recollection of this case is correct, the surgeon did make a diagnosis in spite of what Dr. Brailey has said. It was based largely on the history and the finding of the mass. I thought he had an abscess of the liver, and I therefore made an incision directly over the mass and found it was in the liver, chiefly in the left lobe. I aspirated it with a long needle and discovered a deep-seated abscess. It has been pointed out that liver abscesses are tender and it has been said that if one operates under local anesthesia the exact location of the abscess can be identified by palpation. We have tried that here and have found that most of the liver abscesses are not tender until they present near the surface and set up local peritonitis or pleuritis on the diaphragmatic surface. We drained the abscess, but it was obvious to me even at that time that there were many others. I could feel several suspicious spots throughout the liver.

It was my opinion that the thing started from his appendix. Whether I was influenced by the common history of appendicitis as a seat of the source, I cannot say.

DR. BRAILEY: Would not a more careful history have revealed something?

DR. SWEET: As a matter of fact, he was gone over with a great deal of care in an excellent clinic here in Boston, where the history and examination are notoriously thorough and complete, and they did not make a diagnosis of appendicitis. He was seen by one of our surgeons at that clinic, and he did not make a diagnosis. We questioned the patient at length and thought that there was something going on in the abdomen, with the chills and fever. In a young man who subsequently had some sort of inflammatory reaction in the liver we believed that the most likely source was the appendix. By the time he was first seen, however, the process was obviously beyond the point where an operation on the appendix would have been of any use.

CLINICAL DIAGNOSIS

Liver abscess.

DR. BRAILEY'S DIAGNOSES

Acute suppurative cholangitis.
Pylephlebitis?

ANATOMICAL DIAGNOSES

Appendicitis, acute gangrenous, with rupture into root of mesentery.
Appendiceal abscess.
Pylephlebitis, suppurative: ileocolic and portal veins.
Hepatic abscesses, multiple.
Jaundice.
Splenitis, toxic.
Thrombosis of splenic vein.
Operative wound: drainage of hepatic abscess.

PATHOLOGICAL DISCUSSION

DR. MALLORY: At autopsy we found a liver filled with abscesses. On making a nick in the portal vein, pus welled from it, and then on pressing any spot in the liver more pus could be squeezed out of the portal vein, making it clear that all the multiple abscesses in the liver communicated with the portal system. Tracing down the portal radicles, we found the ileocolic vein likewise filled with pus. In the right lower quadrant was a localized peritoneal abscess, and projecting into that was the stump of an appendix from which the tip had blown off.

The liver weighed 2600 gm., and the spleen 800 gm., so that there was a lot of tissue in the upper abdomen. It is not surprising that the x-rays did not penetrate very well in that region. Our postmortem cultures from these abscesses showed only colon bacilli and streptococci. We were unable to confirm the report of Friedländer's bacillus, which would, I think, be a very unlikely organism to find secondary to a perforated appendix.

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The Journal does not hold itself responsible for statements made by any contributor.

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INSTITUTE ON INDUSTRIAL MEDICINE

Every registered physician in Massachusetts has received a copy of the program of the postgraduate institute, "Industrial Problems in Medical Practice," to be held at the Harvard Club, 374 Commonwealth Avenue, Boston, on Saturday, November 7, under the auspices of the Committee on Postgraduate Instruction and the Committee on Industrial Health, Massachusetts Medical Society, in co-operation with the Massachusetts Department of Public Health and the Division of Occupational Hygiene, Massachusetts Department of Labor and Industries.

This attractive all-day program by authoritative speakers, which appears elsewhere in this issue of the *Journal*, should appeal to every practicing physician, particularly in view of the fact that industrial medicine, in one or another of its many ramifications, is of paramount importance to the continuation of the war effort and a topic with which the majority of physicians are unfamiliar.

Luncheon and dinner will be served at the Harvard Club, but those who plan to attend these functions should signify their intentions in advance, since the seating and serving facilities are limited. Dr. George Leonard Schadt, president of the Society, will preside at the dinner, which will be followed by two serious but entertaining speakers. Because of the timeliness of the subject to be discussed at this meeting, all physicians are urged to attend.

NATIONAL HEARING WEEK

Deafness is a national ill, and now that another war is in process, those so afflicted will increase in alarming numbers. Among the hazards to which the hearing organ may be exposed are excessive noise, gunfire, direct injury and barometric influence. Consequently, there probably has never been a time when both physicians and the laity need to be made more aware of the organized efforts that are being made for the prevention and amelioration of deafness.

The American Society for the Hard of Hearing and its one hundred and twenty-one local affiliates in the United States and Canada have done a great deal to educate and to bring about co-operation among the public, members of the medical profession and the hard of hearing. Among other things, it has accomplished a notable piece of work in fostering hearing tests and promoting lip reading in the public schools, in investigating hearing aids and advertised "cures" and in encouraging research in the medical control of deafness. The society also promotes adult lip reading, social service and vocational opportunities for the hard of hearing everywhere, thus acting as a unifying force to make life easier and better for those so afflicted.

The Boston Guild for the Hard of Hearing, the local chapter, is uniting in the nation-wide observance of National Hearing Week, October 25 to 31. This period will be devoted to the education of the public in the problems of the hard of hearing and to the demonstration of the many facilities that are provided. Because this annual event, held at the headquarters at 283 Commonwealth Avenue, is of so much value to the population as a whole, three papers in this issue of the *Journal* are devoted to the subject of hearing health. Both otologists and general practitioners should give their hearty endorsement to this constructive service for the hard of hearing.

MEDICAL EPONYM

Sahli Hemometer

The hemometer was first described by Professor Herman Sahli (1856-1933), of Bern, in a paper, entitled "Über ein einfaches und exactes Verfahren der klinischen Hämometrie [A Simple and Exact Method of Clinical Hemometry]," which was read at the Twentieth Congress for Internal Medicine in April, 1902. The article appeared in *Verhandlungen des Congresses für Innere Medicin* (**20**:230-234, 1902). A portion of the translation follows:

> After numerous attempts, I have succeeded in finding a method of converting the hemoglobin in a solution of blood into a derivative, by means of a very simple reaction, whereby stable standard solutions can be prepared and colorimetric determinations carried out. . . . The method consists simply in adding to the blood ten times the amount of one-tenth normal hydrochloric acid. After a few seconds, the solution turns deep brown and becomes a clear, brownish yellow after dilution with ordinary water. The pigment content may be colorimetrically determined by means of a similar standard solution, which can be so made as to be completely stable.

R. W. B.

MASSACHUSETTS MEDICAL SOCIETY

COMMITTEES ON POSTGRADUATE INSTRUCTION AND INDUSTRIAL HEALTH

The program for the postgraduate institute, "Industrial Problems in Medical Practice," to be held at the Harvard Club, 374 Commonwealth Avenue, Boston, on Saturday, November 7, is as follows:

Morning Session

Presiding Chairmen: Dr. Dwight O'Hara
Dr. Reginald Fitz

8:30 *Registration.*
9:15 *Opening Remarks.*
9:30 *The Value of Pre-employment Examinations.* Dr. Daniel L. Lynch.
9:45 *The Personality of Industrial Applicants.* Commander A. Warren Stearns, M.C., U.S.N.R.
10:00 *Toxic Fumes and Gases in Industry.* Dr. Alan R. Moritz.
10:45 *Industrial Dermatitis.* Dr. John G. Downing.
11:15 *Question Period.*
11:30 *Round-Table Discussion on the Treatment of Burns.* Drs. Edward D. Churchill, S. Howard Armstrong, Oliver Cope and Charles C. Lund.
12:45 *Luncheon.*

Afternoon Session

Presiding Chairmen: Dr. Bennett F. Avery
Dr. C. Sidney Burwell

2:00 *Injuries to the Eyes.* Lt. Commander Edwin B. Dunphy, M.C., U.S.N.R.
2:30 *Examination and Diagnosis of Lame-Back Conditions.* Dr. Frank R. Ober.
3:15 *Recess.*
3:30 *Round-Table Discussion on the Modern Treatment of Wounds.* Drs. Champ Lyons, Henry C. Marble, Joseph H. Shortell and Gordon M. Morrison.

Evening Session

Presiding Chairman: Dr. George Leonard Schadt

6:30 *Dinner.*
Responsibility of Physicians to Industry in Winning the War. Lt. Colonel Anthony J. Lanza, M.C., U.S.A.
Sterilization in Operating Rooms. Dr. Carl W. Walter.

Reginald Fitz, *Chairman*
Committee on Postgraduate Instruction

Dwight O'Hara, *Chairman*
Committee on Industrial Health

COMMITTEE ON MATERNAL WELFARE

Analysis of Causes of Maternal Death in Massachusetts during 1941

Sepsis

The tabulation printed in last week's issue of the *Journal* showed that 243 puerperal deaths occurred in Massachusetts during 1941 and that, of these, 48 patients died of sepsis. In the septic

cases, 22 were associated with abortion, of which 13 were medicolegal. It is impossible to say how many of these were criminal abortions, but there was only 1 case that might have been associated with a spontaneous abortion. There were 3 cases in which drugs were said to have been the causative factors, although there is no pharmacologic basis for the assumption that drugs play a part in abortions. Thirteen autopsies were performed, and 7 of these were in cases that followed septic abortions, 2 of which were proved to have been associated with gas-bacillus infection.

These 22 septic cases following abortion illustrate the seriousness of attempting to interfere with a normal pregnancy; and the figures, of course, do not include the far greater number of patients who, following an abortion, developed pelvic sepsis with invalidism and, oftentimes, sterility, which not infrequently demanded hysterectomy. A frank discussion of the dangers of abortion is the only way in which to bring the seriousness of this problem to the laity. Of course, all these deaths are preventable.

Of the other 26 cases allocated to sepsis, 6 were associated with normal delivery. One of these may have been caused by alveolar abscesses that were surgically drained. There was not enough evidence to comment on the value of chemotherapy in such cases.

It is gratifying to find that only 3 septic cases were associated with operative vaginal deliveries. Two patients were delivered by low forceps: in one case, sepsis developed immediately after delivery and an autopsy showed that peritonitis had been caused by a rupture of the lower uterine segment, and the other patient developed a fever on the third day after delivery and in spite of apparently intelligent chemotherapy succumbed nineteen days later. The third case in this group was a patient, six and a half months pregnant, in whom a cicatricial cervix made normal dilatation impossible; insertion of a Voorhees bag was attempted unsuccessfully, and sepsis developed intra partum; delivery was consummated by a vaginal cesarean section, and the patient died a few hours later.

Fifteen septic cases were delivered by cesarean section. Of these, 3 had the benefit of autopsy: in two, the diagnosis of peritonitis was confirmed, and in the third, adynamic ileus was apparently the cause of death, without sepsis being the underlying factor. One section was done because of a placenta previa. There were 3 cases in which cesarean sections had been previously performed. The first patient, who had had acute heart failure and chronic bronchitis associated with myocarditis in 1931, was given spinal anesthesia to prevent inhalation irritation; however, sepsis developed. Another patient had no apparent ante-partum infection but developed sepsis immediately following a planned cesarean section. There is nothing of particular interest about the third case.

Of the 2 remaining septic cases, one was proved at autopsy to have had a double uterus and was associated with toxemia, and the other was complicated by a placenta accreta. The latter patient delivered herself of a macerated fetus, but the placenta did not come away. There was no bleeding, the placenta remaining in situ until the day following delivery, when the patient's temperature rose to 103°F. An accreta was diagnosed after an unsuccessful attempt to remove the placenta; hysterectomy was performed, and general peritonitis developed.

It is gratifying that bungling operations played no part in these septic deaths. The handling of the case in which the Voorhees bag was employed unsuccessfully in a patient, six and a half months pregnant, who was finally delivered by vaginal cesarean section after sepsis had developed, is, of course, open to criticism. The case of ruptured uterus associated with sepsis was not appreciated until the autopsy was performed. There were no versions in the entire series.

Three points of particular interest evolve from this study: the small number of cases associated with injudicious operating; the increased number of autopsies over any previous year; and the disturbing fact that fatal sepsis occurred in 3 cases of planned repeat cesarean section, explainable only by inadequate aseptic technic — such deaths are regrettable and should never occur.

DEATHS

BLAKELY — David N. Blakely, M.D., of Brookline, died October 15. He was in his seventy-sixth year.

Born in Campton, New Hampshire, Dr. Blakely attended Dartmouth College. After four years in Aintab, Turkey, he returned to this country and entered Dartmouth Medical School, receiving his degree in 1896. He spent four years at the Boston City Hospital and then practiced in Boston for several years. Dr. Blakely was assistant medical director of the New England Mutual Life Insurance Company at the time of his death. He was a fellow of the Massachusetts Medical Society and the American Medical Association. Dr. Blakely was active in the affairs of the Society for many years, having served as a member of the Council from 1918 to 1940 and as chairman of the Committee on Membership and Finance from 1922 to 1938.

His widow, a daughter, a son, a sister and a brother survive him.

GORMAN — John W. Gorman, M.D., of Brockton, died July 28. He was in his sixty-fifth year.

Dr. Gorman received his degree from the College of Physicians and Surgeons, Boston, in 1900. He was a former member of the Massachusetts Medical Society.

CORRESPONDENCE

THE APPROACHING REFERENDUM

To the Editor: On November 3 the voters of Massachusetts will have an opportunity of deciding at the polls whether physicians are to be allowed to practice their profession in accordance with the best medical standards or whether an old blue law on the statute books of the Commonwealth will continue to stand in the way of their saving the health and even the lives of some of their patients. The referendum to be voted on means just this: duly registered physicians shall be permitted to give contraceptive advice to *married* women whose *health* or *lives* will be jeopardized by pregnancy.

It is an established medical fact that pregnancy imposes a strain on a woman and that in the presence of many diseases (for example, heart disease, nephritis, tuberculosis, certain psychiatric conditions and so forth) continuation of pregnancy or repeated pregnancies may seriously damage the health or even kill some of these women.

It would seem superfluous, therefore, to argue the obvious desirability of this measure, especially before a medical audience, were it not for the devious, obscurantist and fallacious arguments that have been spread as a veritable school of red herrings across the path of truth and that may unfortunately confuse many people; these should be briefly answered.

It is believed by the Catholic Church that such contraceptive methods violate God's will and hence must not be used under any circumstances. This law will in no way interfere with those holding these beliefs; as the state Supreme Court itself has stated, the law is purely permissive, no Catholic physician or patient need use it. It merely extends to those not holding this religious belief a vital medical right, which a minority religious group would withhold from them.

It has been charged that the referendum promotes disunity and thus impairs the war effort. Actually the opposite is the case: true unity and true democracy only exist when the civil and religious liberties of all are protected and mutually respected. It is the opponents of this measure who are sowing disunity by perpetuating intolerance, and they are trying to maintain an economically undemocratic situation whereby the poor people of the State, who can only be adequately cared for in clinics, are deprived of medical help, which many of the well-to-do are now obtaining in one way or another.

The charge that this law will increase license and promiscuity is equally absurd, and is by implication a slur on the honor of the medical profession. Since it will apply only to married women for health reasons, any advice given to any other women will have no more legal sanction than exists at present. The hypocrisy of those making this charge is evident from the fact that they have never in concerted fashion publicly opposed the sale of condoms and similar articles which, it is well known, are the contraceptives chiefly used in sexual promiscuity; their sale has been declared legal because they can be sold ostensibly "for the prevention of disease." This measure will improve moral standards because it will give married women who need it the right to obtain, and physicians the right to prescribe, this advice *legally*.

Another accusation is that this law will lead to race suicide. It is patent that the number of healthy children that the women who will come under this law will fail to produce is extremely small, and will be more than overbalanced by the increased number of viable children who will be born when sick mothers can properly space their children and limit the size of their families. Statistics prove our point: for example, in Ireland, where birth control has long been outlawed by church and state, the population has been cut in half during the last one hundred years, the most catastrophic birth decline of any nation in the world; on the other hand, in Holland, which pioneered in public birth-control clinics in 1880, the population has doubled in 60 years, owing to a much higher marriage rate and very low infant and maternal mortality rates. Other factors than the legality of contraceptive advice determine birth rates.

We often think with pride that Massachusetts ranks high in the medical world. But in this case all states in the Union except Massachusetts and Connecticut accord doctors the right to give needed advice. The challenge has been thrown down to the people of Massachusetts, and especially to physicians. It is an issue that far transcends the particular point with which this referendum deals. The issue is, Shall doctors be denied the right to practice their profession according to the highest medical standards and in accordance with the dictates of their own consciences?

WILLIAM H. ROBEY, M.D., *Chairman*
LAURENCE B. ELLIS, M.D., *Secretary*
Physicians Committee for the Defense of Medical Rights
143 Newbury Street
Boston

NOTICES

ANNOUNCEMENTS

DR. HENRY R. VIETS announces the removal of his office to 262 Beacon Street, Boston, on November 1.

The Bacteriological Laboratory of the Health Department of the City of Boston has been transferred from Room 1101, City Hall Annex, to new quarters in the Haymarket Square Relief Building, where the entire personnel of the Health Department will shortly be located. The Bacteriological Laboratory can be reached by the same telephone numbers, and maintains the same bacteriologic and serologic services, with the advantage of modern facilities and equipment.

MASSACHUSETTS GENERAL HOSPITAL

A meeting of the Hospital Research Council will be held in the Bigelow Amphitheater of the White Building on Tuesday, October 27, at 5 p.m.

PROGRAM

Some New Data on Oxygen Poisoning. Drs. C. M. Williams and H. K. Beecher.

Notes on the Action of Colchicine. Dr. Austin M. Brues.

The Relation of Vascular Disease to the Hypertensive State based on a Study of Renal Biopsies from 100 Hypertensive Patients. Drs. Benjamin Castleman and R. H. Smithwick.

17-Ketosteroids Excretion in Normal and Abnormal Children. Drs. N. B. Talbot and A. M. Butler.

(Notices continued on page viii)